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TRANSACTIONS

OF THE

COLLEGE OF PHYSICIANS

OF

PHILADELPHIA

THIRD SERIES

VOLUME THE FORTY-NINTH

PHILADELPHIA
PRINTED FOR THE COLLEGE
1927

NOTICE

The present volume of TRANSACTIONS contains the papers read before the College from January, 1927, to December, 1927, inclusive.

The Committee of Publication thinks it proper to say that the College holds itself in no way responsible for the statements, reasonings, or opinions set forth in the various papers published in its TRANSACTIONS.

EDITED BY

WALTER G. ELMER, M.D.

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DORNAN, PRINTER

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LIST

OF THE

PRESIDENTS OF THE COLLEGE FROM THE TIME OF ITS INSTITUTION

ELECTED	
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1805	WILLIAM SHIPPEN
1809	ADAM KUHN
1818	THOMAS PARKE
1835	THOMAS C. JAMES[1]
1835	THOMAS T. HEWSON
1848	GEORGE B. WOOD
1879	W. S. W. RUSCHENBERGER
1883	ALFRED STILLÉ
1884	SAMUEL LEWIS[2]
1884	J. M. DA COSTA
1886	S. WEIR MITCHELL
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1892	S. WEIR MITCHELL
1895	J. M. DA COSTA
1898	JOHN ASHHURST, JR.
1900	W. W. KEEN
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1922	THOMAS R. NEILSON
1925	HOBART A. HARE

[1] Died four months after his election.
[2] Resigned on account of ill-health.

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1927

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FELLOWS

OF THE

COLLEGE OF PHYSICIANS OF PHILADELPHIA

DECEMBER, 1927

* Non-resident Fellows. † Fellows who have commuted dues. ‡ Deceased.

ELECTED

1892. Abbott, Alexander Crever, M.D., Sc.D., Dr. P.H., Professor of Hygiene and Bacteriology and Director of the School of Hygiene and Public Health, University of Pennsylvania. 4016 Pine St.

1912. Addison, William H. F., A.B., M.D., Professor of Normal Histology and Embryology in the University of Pennsylvania. Medical Laboratories, University of Pennsylvania.

1926. Adler, Francis Heed, M.A., M.D., Assistant Surgeon, Wills Eye Hospital; Instructor in Ophthalmology and Physiology, University of Pennsylvania. 313 S. Seventeenth St.

1905. Adler, Lewis H., Jr., M.D., Formerly Professor of Proctology in the Graduate School of Medicine, University of Pennsylvania; formerly Prosector to the Professor of Anatomy in the University of Pennsylvania; Consulting Surgeon to the Charity Hospital. 313 S. Seventeenth St.

1914. Aiken, Thomas Gerald, M.D., Physician-in-Chief to the Chester County Hospital, West Chester, Pa.; Pathologist to the Country Branch of the Rush Hospital for Tuberculosis. Berwyn, Pa.

1913. Alexander, Emory G., M.D., Surgeon to St. Christopher's Hospital for Children; Associate Surgeon to the Episcopal Hospital; Clinical Professor of Surgery in the Woman's Medical College of Pennsylvania; Demonstrator of Fracture Dressings in the Jefferson Medical College, and Assistant Surgeon to the Kensington Hospital for Women. 337 S. Eighteenth St.

ELECTED

1906. Allen, Francis Olcott, Jr., A.B., M.D. 2216 Walnut St.

1896. Allyn, Herman B., A.B., M.D., Visiting Physician to the Philadelphia General Hospital and President of the Medical Board. 3910 Chestnut St.

1888. Anders, James M., M.D., LL.D., Professor of Medicine and Clinical Medicine in the Medico-Chirurgical College; Officer de l'Instruction Publique. 250 S. Seventeenth St.

1905. Anspach, Brooke M., M.D., Professor of Gynecology in the Jefferson Medical College; Attending Gynecologist, Jefferson Hospital; Attending Gynecologist to the Bryn Mawr Hospital. 1827 Spruce St.

1905. Appleman, Leighton F., M.D., Attending Surgeon to the Wills Eye Hospital; Associate Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Ophthalmologist to the Frederick Douglass Memorial Hospital and to the Burd School; Demonstrator of Pharmacy and Materia Medica, and Instructor in Therapeutics in the Jefferson Medical College. 308 S. Sixteenth St.

1922. Arnett, John Hancock, A.B., M.D., Instructor in Medicine, Medical School, University of Pennsylvania; Assistant in Medicine and Students' Physician, University Hospital. 2116 Pine St.

1906. Ashhurst, Astley Paston Cooper, A.B., M.D., Professor of Clinical Surgery, University of Pennsylvania; Surgeon to the Episcopal Hospital and to the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases. 257 S. Sixteenth St.

1893. Ashton, Thomas G., M.D., Physician to the Philadelphia General Hospital. Wynnewood, Pa.

1920. Astley, George Mason, M.D., Assistant Surgeon, Samaritan Hospital. 5317 Master St.

1914. Austin, J. Harold, B.S. (Univ. of Penna.), M.D., Professor of Research Medicine, University of Pennsylvania. John Herr Musser Department of Research Medicine, University of Pennsylvania.

1906. Babbitt, James A., A.B. (Yale), A.M. (Haverford), M.D., Assistant Professor of Otology, University of Pennsylvania; Associate Professor of Oto-Laryngology, Graduate School of Medicine, University of Pennsylvania; Assistant Laryngologist and Aurist, Lankenau Hospital; Laryngologist, Mary Drexel Children's Hospital. 1912 Spruce St.

ELECTED

1910. Baer, Benjamin F., Jr., B.S., M.D., Associate Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Assistant Ophthalmologist, University Hospital; Attending Surgeon to Wills Eye Hospital; Ophthalmologist, Howard Hospital. 1927 Spruce St.

†1892. Baker, George Fales, B.S., M.D. 1818 Spruce St.

1911. Baldwin, James Harvey, A.B., M.D., Surgeon to the Methodist Hospital. 1426 Pine St.

1889. Baldy, John Montgomery, M.D., Surgeon to the Gynecean Hospital; Consulting Surgeon to the Jewish and the Frederick Douglass Memorial Hospitals. Devon, Pa.

1916. Balentine, Percival L., M.D., Assistant Surgeon, Wills Eye Hospital; Demonstrator of Ophthalmic Surgery in the Philadelphia Polyclinic. 302 Weightman Building, 1524 Chestnut St.

1898. Balliet, Tilghman M., A.M., M.D., Professor of Therapeutics at Dartmouth College, Hanover, N. H.; Physician to the Old Man's Home. 3920 Sansom St.

1911. Barnard, Everett P., M.D., Obstetrician to the Maternity Hospital; Assistant Instructor in Obstetrics in the University of Pennsylvania. 1820 S. Rittenhouse Square.

1924. Bartle, Henry J., B.S., M.D., Clinical Assistant, Department for Diseases of the Stomach and Bowels, Jefferson Medical College and Hospital. 501 Medical Arts Bldg.

1921. Bauer, Edward L., M.D., Professor of Children's Diseases, Jefferson Medical College; Pediatrist to the Jefferson and Memorial Hospitals and the Philadelphia Hospital for Contagious Diseases; Consulting Pediatrist, Germantown Hospital. 345 S. Nineteenth St.

1883. Baum, Charles, A.M., M.D., Ph.D. 1828 Wallace St.

1922. Bazett, Henry Cuthbert, M.A., M.D. (Oxon.), F.R.C.S. (England), Professor of Physiology, University of Pennsylvania. Bryn Mawr, Pa.

1908. Beardsley, Edward J. G., M.D., L.R.C.P. (Lond.), Clinical Professor of Medicine in the Jefferson Medical College; Chief Clinical Assistant in the Out-patient Medical Department of the Jefferson Medical College Hospital; Assistant Physician to the Jefferson Hospital. 1919 Spruce St.

*1916. Bell, William Hemphill, M.D., Captain, Medical Corps, U. S. Navy, Commanding Officer, U. S. Naval Hospital, Newport, R. I. 7 Cottage St., Newport, R. I.

ELECTED

1923. Bergey, David H., A.M., M.D., B.S., Dr. P.H., Professor of Hygiene and Bacteriology, University of Pennsylvania. 206 S. Fifty-third St.

*1903. Biggs, Montgomery H., M.D., Surgeon to the Rutherford Hospital; Chief Surgeon of the Carolina, Churchfield and Ohio Railway. Rutherfordton, N. C.

1918. Billings, Arthur E., M.D., Clinical Assistant in Surgery, Dept. B, Jefferson Medical College; Surgeon to Pennsylvania and Bryn Mawr Hospitals. 2020 Spruce St.

1917. Birdsall, Joseph C., A.B., A.M., M.D., Urologist, Polyclinic Section of the Medico-Chirurgical College and Hospital, Graduate School of Medicine, University of Pennsylvania; Assistant in Genito-urinary Dispensary, Presbyterian Hospital; Instructor in Surgery, University of Pennsylvania. 4610 Spruce St.

1908. Bland, Pascal Brooke, M.D., Professor of Obstetrics, Jefferson Medical College; Visiting Obstetrician, Jefferson Medical College; Gynecologist to St. Joseph's Hospital. 1621 Spruce St.

1917. Block, Frank B., A.B., M.D., Surgeon to the Jewish Hospital; Instructor in Gynecology in the University of Pennsylvania. Medical Arts Building.

1894. Bochroch, Max H., M.D., Professor of Nervous and Mental Diseases, Temple University; Visiting Physician to the Psychopathic Wards of the Philadelphia General Hospital. 1539 Pine St.

1896. Boger, John A., A.M., M.D., Surgeon to St. Mary's Hospital; Surgeon to the Stetson Hospital; Surgeon to the Dispensary of the Episcopal Hospital. 2213 N. Broad St.

1923. Bond, Earl D., A.B., M.D., Physician-in-Chief, Pennsylvania Hospital, Department for Mental and Nervous Diseases; Professor of Psychiatry, Graduate School of Medicine, University of Pennsylvania. Bryn Mawr, Pa.

1911. Bonney, Charles W., A.B., M.D., Associate in Topographic and Applied Anatomy in the Jefferson Medical College. 1117 Spruce St.

1924. Boon, David J., M.D., Opthalmologist, Memorial Hospital and Philadelphia Hospital for Contagious Diseases; Assistant Ophthalmic Surgeon, Episcopal Hospital. 1532 N. Fifteenth Street.

1921. Borzell, Francis Frank, M.D., Roentgenologist, Frankford Hospital; Chief Clinical Assistant, Roentgen Depart-

ELECTED

ment, Jefferson Medical College; X-Ray Consultant, State Hospital for the Insane, Norristown, Pa. 4910 Frankford Ave.

1913. Boston, L. Napoleon, A.M., M.D., Professor of Physical Diagnosis, Graduate School, University of Pennsylvania; Physician to the Philadelphia General and Northwestern General Hospitals. 1737 Chestnut St.

1919. Bowen, David Ralph, M.D., Radiologist to the Pennsylvania Hospital. 82 W. Lacrosse Ave., Lansdowne, Pa.

1923. Bower, John O., M.D. 2008 Walnut St.

1891. Boyd, George M., M.D., Professor of Obstetrics, Graduate School of Medicine, University of Pennsylvania; Consultant to the Philadelphia Lying-in Charity; Consulting Obstetrician to the Preston Retreat; Obstetrician and Gynecologist to the Medico-Chirurgical Hospital. 1909 Spruce St.

1907. Boyer, Henry Percival, M.D., Neurologist to the Stetson Hospital; Physician to the Philadelphia Home for Incurables; Assistant Physican to the Orthopædic Hospital and Infirmary for Nervous Diseases. 4602 Baltimore Ave.

1925. Boykin, Irvine M., M.D., Associate Surgeon to Episcopal Hospital; Instructor in Surgery, Graduate School of Medicine, University of Pennsylvania. Elkins Park, Pa.

1907. Bradley, William N., Ph.G., M.D., Visiting Pediatrist to Howard Hospital; Visiting Pediatrist to Philadelphia General Hospital; Associate in Pediatrics, School of Medicine, University of Pennsylvania; Assistant Professor of Pediatrics, Graduate School of Medicine, University of Pennsylvania. 1725 Pine St.

1921. Bransfield, John W., M.D., Clinical Professor of Surgery, Woman's Medical College; Visiting Surgeon to St. Agnes, St. Vincent's and Woman's College Hospitals. 2025 Walnut Street.

1903. Branson, Thomas F., M.D., Attending Physician to the Bryn Mawr Hospital. Rosemont, Pa.

1891. Brinton, Lewis, M.D., Physician-in-Chief to the American Hospital for Diseases of the Stomach. 1301 Medical Arts Bldg., Sixteenth and Walnut Sts.

1900. Brinton, Ward, A.M., M.D., Chief of Division of Tuberculosis, Department of Public Health of Philadelphia; Clinical Professor of Medicine, Woman's Medical College of Pennsylvania; Visiting Physician, Department for Tuberculosis, Philadelphia General Hospital, and Eaglesmere Sanatorium. 1423 Spruce St.

ELECTED

1917. Bromer, Ralph S., A.B. (Yale), M.D., Roentgenologist to the Episcopal and the Orthopædic Hospitals. 504 W. Mt. Airy Ave.

1919. Brown, Henry P., Jr., M.D., Surgeon to Pennsylvania and Children's Hospitals; Assistant Surgeon, Presbyterian Hospital. 624 Medical Arts Building.

1916. Brown, Samuel Horton, M.D. 1901 Mt. Vernon St.

1887. Brubaker, Albert P., A.M., M.D., Emeritus Professor of Physiology and Medical Jurisprudence in the Jefferson Medical College. 3426 Powelton Ave.

1916. Buckley, Albert Coulson, M.D., Medical Superintendent, Friends' Hospital, Frankford; Associate Professor of Psychiatry, Graduate School of Medicine, University of Pennsylvania. Frankford, Phila.

1927. Burns, Michael A., M.D., Assistant Professor Nervous and Mental Diseases, Jefferson Medical College; Chief of the Nervous and Mental Clinic, Jefferson Hospital; Visiting Neurologist, Philadelphia General Hospital; Neuropsychiatrist, St. Mary's Hospital. N. E. Cor. Twentieth and Chestnut Sts.

1906. Burns, Stillwell C., M.D., Associate Professor of Surgery in the Graduate School of Medicine, University of Pennsylvania. 1925 Spring Garden St.

1892. Burr, Charles W., M.D., Professor of Mental Diseases in the University of Pennsylvania; Neurologist to the Philadelphia General Hospital. 1918 Spruce St.

1906. Butler, Ralph, M.D., Professor of Diseases of the Nose and Throat in the Philadelphia Polyclinic and the College for Graduates in Medicine; Chief of the Dispensary for Diseases of the Ear at the University Hospital; Assistant Professor of Otology in the University of Pennsylvania; Laryngologist and Aurist to the Lankenau Hospital. 2007 Chestnut St.

*1908. Cadbury, William W., A.M., M.D., Canton Christian College, Canton, China.

†1907. Cadwalader, Williams B., M.D., Assistant Professor of Neurology, School of Medicine, University of Pennsylvania; Neurologist to the Presbyterian Hospital; Consulting Neurologist to the Bryn Mawr Hospital. 1206 Medical Arts Building.

1905. Carmany, Harry S., M.D., Surgeon to Memorial Hospital, Roxborough; Associate Surgeon of the Episcopal

ELECTED

Hospital; Surgeon to the Dispensary of the Episcopal Hospital. 366 Green Lane, Roxborough.

1924. Carncross, Horace, M.D., 1003 Spruce St.

1910. Carnett, John Berton, M.D., Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Associate in Surgery, Medical Department, University of Pennsylvania; Surgeon to Philadelphia General Hospital; Chief Surgeon to the American Stomach Hospital. 2012 Spruce St.

1905. Carpenter, Herbert B., M.D. 1805 Spruce St.

1926. Carpenter, Howard Childs, M.D., Vice-Dean for Pediatrics and Professor of Pediatric Hygiene, Graduate School of Medicine, University of Pennsylvania; Assistant Professor of Pediatrics, School of Medicine, University of Pennsylvania; Director, Department for the Prevention of Disease, Children's Hospital. 1805 Spruce St.

1895. Carpenter, John T., M.D., Attending Ophthalmologist to the Bryn Mawr Hospital. 2030 Chestnut St.

1917. Carson, John B., M.D., Physician to Episcopal Hospital; Assistant Physician, Pennsylvania Hospital. 1802 Pine Street.

1922. Case, Eugene A., M.D., Professor of Morbid Anatomy, Graduate School of Medicine, University of Pennsylvania; Assistant Pathologist, Philadelphia General Hospital. Lansdowne, Pa.

1892. Cattell, Henry W., A.M., M.D. 1806 Spruce St.

1900. Chance, Burton, M.D., Attending Surgeon to the Wills Eye Hospital; Consulting Ophthalmologist Eastern Pennsylvania Institution for the Feeble-minded and Epileptics; Ophthalmic Surgeon to the Pennsylvania Railroad Company. 315 S. Fifteenth St.

1897. Cheston, Radcliffe, M.D., Visiting Physician to the Chestnut Hill Hospital; Consulting Physician to the Germantown Hospital and to the Pennsylvania Institution for the Deaf and Dumb. Chestnut Hill.

1903. Chrystie, Walter, M.D. One of the Senior Physicians to Bryn Mawr Hospital. Bryn Mawr, Pa.

‡1899. Clark, John G., M.D., Professor of Gynecology in the University of Pennsylvania; Gynecologist-in-Chief to the University Hospital. 2017 Walnut St.

1926. Clarke, J. Alexander, A.B., M.D., Chief Clinical Assistant, Out-Patient Department of Applied Immunology, Jefferson

ELECTED

Hospital; Assistant Physician, Philadelphia General Hospital; Instructor in Medicine, Undergraduate School, University of Pennsylvania. 334 S. Twenty-first St.

1926. Clerf, Louis H., M.D., Instructor in Bronchoscopy and Esophagoscopy and Demonstrator of Laryngology, Jefferson Medical College; Assistant Professor of Bronchoscopy and Esophagoscopy, Graduate School of Medicine, University of Pennsylvania; Chief Clinical Assistant in Bronchoscopy and Esophagoscopy, Jefferson Hospital; Assistant in Bronchoscopy and Esophagoscopy, University Hospital. The Esmond, Twelfth and Spruce Sts.

1910. Cloud, J. Howard, M.D., Attending Physician to the Bryn Mawr Hospital. 11 W. Montgomery Ave., Ardmore, Pa.

1903. Coates, George Morrison, A.B., M.D., Professor of Otology, Graduate School of Medicine, University of Pennsylvania; Associate Surgeon, Ear, Throat and Nose, Pennsylvania Hospital; Consulting Laryngologist to the Philadelphia Orphanage and to the Sharon Hospital, Sharon, Conn. 1721 Pine St.

1908. Codman, Charles A. E., M.D., Physician to the American Oncologic Hospital. 4116 Spruce St.

1922. Cohen, Leon Solis, A.B., M.D., Clinical Assistant of Roentgenology, Jefferson Medical College. 1923 Spruce St.

1907. Cohen, Myer Solis, A.B., M.D., Assistant Professor in Medicine, Graduate School of Medicine, University of Pennsylvania; Lecturer on Medical Jurisprudence, Woman's Medical College of Pennsylvania; Director of the Jules E. Mastbaum Research Laboratory of the Jewish Hospital; Pediatrist to the Jewish Hospital. 1833 Pine St.

1888. Cohen, Solomon Solis, M.D., Emeritus Professor of Clinical Medicine in the Jefferson Medical College; Emeritus Professor of Medicine and Therapeutics, Philadelphia Polyclinic and College for Graduates in Medicine; Consulting Physician to the Jewish Hospital and to the Rush Hospital. 1906 Walnut St.

1920. Cole, Charles J., A.B., M.D., Visiting Physician, Philadelphia Hospital for Contagious Diseases; Assistant Pediatrist, Polyclinic Hospital; Physician to Out-patient Department, Mary Drexel Home; Assistant Physician to Out-patient Department, Children's Hospital, University Hospital. Elkins Park, Pa.

1898. Coles, Stricker, M.D. Bryn Mawr, Pa.

ELECTED

*1901. Coley, Thomas Luther, A.B., M.D., Major, Medical Corps, U. S. Army. Fortress Monroe, Va.

1903. Coplin, W. M. L., M.D., Professor of Pathology in the Jefferson Medical College; Pathologist to and Director of the Laboratories of the Jefferson Medical College Hospital; Pathologist to the Philadelphia General Hospital and to the Friends' Asylum for the Insane, Frankford; Bacteriologist to the Pennsylvania State Board of Health. Albany Apartments, Atlantic City, N. J.

1912. Copp, Owen, A.B., M.D., Consultant for the Future Development of the Pennsylvania Hospital. 4401 Market St.

1911. Cornell, Walter Stewart, A.B., M.D., Director of Medical Inspection of Public Schools of the City of Philadelphia; Chief of Medical Staff of the House of Detention; Lecturer on Osteology in the University of Pennsylvania. 1919 Cherry St.

1914. Corson, Edward Foulke, M.D., Assistant Professor of Dermatology, Jefferson Medical College; Dermatologist, Children's Hospital of Philadelphia. Medical Arts Bldg.

1907. Council, Malcolm S., M.D., Attending Physician to the Bryn Mawr Hospital; Attending Physician to the Cathcart Home at Devon. Bryn Mawr, Pa.

1924. Cowan, Alfred, M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Assistant Instructor in Ophthalmology, School of Medicine, University of Pennsylvania; Ophthalmologist, Philadelphia General Hospital. 2007 Pine St.

1904. Craig, Frank A., M.D., Instructor in Medicine in the University of Pennsylvania; Visiting Physician to the Henry Phipps Institute, University of Pennsylvania; Visiting Physician to the White Haven Sanatorium; Tuberculosis Consultant to the Presbyterian Hospital. 2011 Pine Street.

1907. Crampton, George S., M.D., Attending Surgeon to the Eye Department of the Pennsylvania Hospital and the Philadelphia Hospital for Contagious Diseases; Assistant Surgeon to the Wills Hospital; Lecturer on Physiologic Optics in the Philadelphia Polyclinic and School for Graduates in Medicine; Ophthalmologist to the Philadelphia Orphanage. 1819 Walnut St.

1924. Crawford, Baxter Lindsey, M.D., Associate in Pathology, Jefferson Medical College; Chief of Laboratory Service,

ELECTED

Jefferson Hospital; Assistant Pathologist, Philadelphia General Hospital. Jefferson Hospital, Tenth and Sansom Sts.

1920. Creighton, William J., B.A., M.D., Assistant Ophthalmic Surgeon, Germantown Hospital and Dispensary; Assistant Dispensary Surgeon, Department Ophthalmology, University Hospital; Instructor in Ophthalmology, University of Pennsylvania Medical School. 258 S. Eighteenth St.

1920. Cross, George Howard, M.D., Ophthalmologist to the Chester Hospital; Instructor in Ophthalmology, Graduate School of Medicine, University of Pennsylvania. Chester, Pa.

1917. Cross, Sumner H., A.B., M.D., Physician to the Abington Memorial Hospital, Abington, Pa. 412 York Road, Jenkintown, Pa.

1921. Crossan, Edward T., M.D., Associate Surgeon, Episcopal Hospital; Associate in Surgery, University of Pennsylvania; Assistant Surgeon Orthopædic Hospital. 257 S. Sixteenth Street.

*1924. Crowell, Bowman C., A.B., M.D., C.M., Associate Director, American College of Surgeons; Director of Clinical Research. 40 E. Erie St., Chicago, Ill.

1904. Cruice, John M., M.D., Physician to St. Agnes and St. Joseph's Hospitals and to the Henry Phipps Institute of the University of Pennsylvania; Instructor in Medicine in the University of Pennsylvania. 2021 Pine Street.

*1910. Cummins, W. Taylor, M.D., Pathologist to the Southern Pacific Hospital; Director of the Mary W. Harriman Research Laboratory, San Francisco, Calif.

1902. Currie, Charles A., M.D., Physician to the Germantown Hospital. West Walnut Lane, Germantown.

1896. Da Costa, John Chalmers, M.D., Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College; Surgeon to the Philadelphia General and St. Joseph's Hospital. 2045 Walnut St.

1887. Daland, Judson, M.D., Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. 317 S. Eighteenth St.

1919. Davies, John R., Jr., M.D., Chief to the Out-patient Nose, Throat and Ear Department of the Pennsylvania, Bryn Mawr and Chestnut Hill Hospitals. 302 S. Nineteenth Street.

ELECTED

1896. Davis, Charles N., M.D., Dermatologist to the Pennsylvania Hospital; Consulting Dermatologist to St. Agnes' Hospital; Assistant Physician to the Dispensary for Skin Diseases in the Howard Hospital. 1931 Spruce St.

1916. Davis, Warren B., M.D., Rhinologist, Laryngologist, and Otologist, St. Agnes' Hospital; Instructor in Surgery, Jefferson Medical College; Assistant Surgeon, Frankford Hospital. 135 S. Eighteenth St.

1900. Davisson, Alex. Heron, M.D. 4514 Springfield Ave.

1894. Deaver, Harry C., M.D., Professor of Surgery in the Woman's Medical College of Pennsylvania; Surgeon to the Episcopal Hospital and to the Children's Hospital of the Mary J. Drexel Home; Surgeon-in-Chief to the Kensington Hospital for Women. 337 S. Eighteenth St.

1887. Deaver, John B., M.D., D.Sc., LL.D., Visiting Surgeon, Hospital of the University of Pennsylvania; Surgeon-in-Chief to the Lankenau Hospital. 1830 DeLancey St.

1902. Dehoney, Howard, M.D. 240 S. Thirteenth St.

1924. Deichler, L. Waller, M.D., Assistant Surgeon, Wills Hospital; Instructor Ophthalmology, University of Pennsylvania; Ophthalmologist, Philadelphia General Hospital. 1737 Chestnut St.

1923. De Long, Perce, M.D. 1904 Chestnut St.

1885. Dercum, Francis X., A.M., M.D., Ph.D., Sc.D., President, American Philosophical Society, Professor Emeritus of Nervous and Mental Diseases in the Jefferson Medical College; Foreign Corresponding Member of the Neurological Society of Paris; Honorary Member of the Psychiatric and Neurological Society of Vienna; Corresponding Member of the Neurological Section of the Royal Society of Medicine; Chevalier de l'Ordre National de la Légion d'Honneur. 1719 Walnut St.

1912. Dewey, J. Hiland, Ph.B., M.D., Assistant Surgeon to Wills Hospital; Ophthalmic Surgeon to St. Francis' Hospital, Trenton, N. J. 1436 Diamond St.

*1911. Dickson, Frank D., M.D. 1807 Federal Reserve Bank Building, Kansas City, Mo.

1908. Dillard, Henry K., Jr., M.D., Physician to the Out-patient Department of the Pennsylvania Hospital; Physician to the Dispensary of the Mary J. Drexel Home. 234 S. Twentieth St.

ELECTED

1924. Dillon, Edward S., A.B., M.D., Assistant Physician, Pennsylvania Hospital; Assistant Instructor in Medicine, School of Medicine, University of Pennsylvania. 2040 Pine St.

1920. Diven, John, A.B., M.D., Pediatrician to St. Christopher's Hospital and Babies' Hospital of Philadelphia. 326 S. Nineteenth Street.

1920. Doane, Joseph Chapman, M.D., Superintendent and Medical Director, Philadelphia General Hospital; Associate Professor of Internal Medicine, Graduate School of Medicine, University of Pennsylvania. Philadelphia General Hospital.

*1897. Dorland, W. A. Newman, A.M., M.D., Professor of Gynecology in the Post-graduate Medical School of Chicago; Professor of Obstetrics in the Chicago College of Medicine and Surgery; Visiting Obstetrician to Cook County Hospital. 185 N. Wabash Ave., Chicago, Ill.

1907. Dorrance, George Morris, M.D., Professor of Maxillo-Facial Surgery, Thomas Evans Institute, University of Pennsylvania; Professor of Surgery and Clinical Surgery, Woman's Medical College; Surgeon in Chief, Woman's College Hospital; Surgeon to St. Agnes' Hospital. 2025 Walnut St.

1902. Downs, Robert N., Jr., M.D., Surgeon to the Dispensary of the Germantown Hospital. 6008 Greene St., Germantown.

1910. Drayton, William, Jr., M.D., Physician to the Philadelphia Hospital for Contagious Diseases; Physician to the Outpatient Department of the Pennsylvania Hospital; Physician to the Pennsylvania Institute for the Instruction of the Blind; Assistant Physician to the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases. 1820 Pine Street.

1911. Earnshaw, Henry Culp, M.S., M.D., Attending Physician to the Hospital of the Good Shepherd, Rosemont; Assistant Attending Physician to the Bryn Mawr Hospital; Attending Physician to the Bryn Mawr Children's Hospital; Pennsylvania Railroad Surgeon. Bryn Mawr, Pa.

*1887. Edwards, William A., M.D., Professor of Pediatrics in the Medical Department of the University of California. R. D. 2, Box 105, El Cajon, Calif.

1924. Egbert, Seneca, M.D., Professor of Hygiene, University of Pennsylvania. Wayne, Pa.

1911. Eliason, Eldridge L., B.A., M.D., Sc.D., Associate in Surgery in the University of Pennsylvania; Surgeon to the Howard

ELECTED

Hospital; Assistant Surgeon to the University of Pennsylvania Hospital, the Philadelphia General Hospital and the American Stomach Hospital. 326 S. Nineteenth St.

1904. Elmer, Walter G., B.S., M.D., Associate Professor of Orthopædic Surgery, Graduate School of Medicine, University of Pennslvania; Clinical Professor of Orthopædic Surgery in the Woman's Medical College of Pennsylvania; Orthopædic Surgeon to the Polyclinic Hospital and to the Philadelphia General Hospital. 1801 Pine St.

1896. Ely, Thomas C., A.M., M.D. 3912 Chestnut St.

1893. Eshner, Augustus A., M.D., Consulting Physician to Mercy Hospital. 1019 Spruce St.

*1905. Evans, Joseph S., Jr., A.B., M.D., Professor of Medicine, Medical School, University of Wisconsin; Physician to State of Wisconsin General Hospital; Consulting Physician, Madison General and St. Mary's Hospitals. 1300 University Ave., Madison, Wis.

1905. Evans, William, M.D. 500 W. Gay St., West Chester.

1912. Eves, Curtis C., M.D., Otolaryngologist to Out-patient Department of the Pennsylvania Hospital; Attending Otolaryngologist to Bryn Mawr Hospital. 1910 Spruce St.

1922. Farley, David L., B.S., M.D., Associate in Medicine, University of Pennsylvania; Physician to Pennsylvania Hospital; Chief of Out-Patient Medical Clinic, Pennsylvania Hospital. 1725 Pine St.

†1903. Farr, Clifford B., A.M., M.D., Director of Laboratories, Pennsylvania Hospital, Department for Mental and Nervous Diseases. Bryn Mawr, Pa.

1893. Farr, William W., M.D., Physician to the Leamy Home. 121 Maplewood Ave., Germantown.

1884. Fenton, Thomas H., M.D. 1319 Spruce St.

1907. Fetterolf, George, A.B., M.D., Sc.D., Professor of Otolaryngology, University of Pennsylvania. 2010 Spruce St.

1924. Fewell, Alexander G., M.D. 1924 Pine St.

1907. Fife, Charles A., A.B., M.D., Associate in Pediatrics in the University of Pennsylvania; Pediatrist to the Presbyterian Hospital; Physician to the St. Christopher's Hospital for Children; Assistant Physician to the Philadelphia General Hospital. 2104 Spruce St.

1884. Fisher, Henry M., A.B., M.D. Jenkintown, Pa.

ELECTED

1910. Fisher, John Monroe, M.D., Associate Professor of Gynecology in the Jefferson Medical College; Gynecologist to the Philadelphia, St. Agnes' and Pottstown Hospitals; Assistant Gynecologist to the Jefferson Medical College Hospital. 345 S. Nineteenth St.

1888. Flick, Lawrence F., M.D. 736 Pine St.

1916. Forst, John R., M.D. 166 W. Coulter St., Germantown.

1908. Foulkrod, Collin, M.D., Obstetrician to the Presbyterian Hospital; Assistant Demonstrator of Obstetrics in Jefferson Medical College. 3910 Chestnut St.

1908. Fox, Herbert, M.D., Director of the William Pepper Clinical Laboratory and Professor of Comparative Pathology, University of Pennsylvania; Pathologist to the Laboratory of Comparative Pathology of the Zoölogical Society of Philadelphia; Pathologist to the Rush Hospital. Haverford, Pa.

†1885. Fox, Joseph M., M.D. Torresdale, Pa.

1906. Fraley, Frederick, Jr., A.B., M.D. Overbrook, Pa.

1897. Frazier, Charles H., A.B., M.D., Sc.D., John Rhea Barton Professor of Surgery, University of Pennsylvania. 133 S. Thirty-sixth St.

1916. Funk, Elmer Hendricks, M.D., Clinical Professor of Medicine and Therapeutics in the Jefferson Medical College; Assistant Physician to the Pennsylvania Hospital; Visiting Physician to the White Haven Sanatorium. 1318 Spruce St.

1899. Gamble, Robert, G. M.D., one of the Attending Physicians to the Bryn Mawr Hospital. Haverford, Pa.

1912. Gaskill, Henry Kennedy, M.D. S. E. Cor. Nineteenth and Spruce Sts.

*1913. Gates, Nathaniel, M.D. 10 Peterboro Ave., Detroit, Mich.

1917. Gerhard, Arthur Howell, M.D. 1615 N. Delaware Ave.

1902. Ghriskey, Albert A., M.D. 3936 Walnut St.

1899. Gibbon, John H., M.D., Professor of Surgery in the Jefferson Medical College; Surgeon to the Pennsylvania Hospital and Consulting Surgeon to the Bryn Mawr Hospital. 1608 Spruce St.

1920. Gill, A. Bruce, A.B., M.D., Professor of Orthopædic Surgery, University of Pennsylvania; Surgeon to the Orthopædic Hospital; Orthopædic Surgeon to the Presbyterian Hospital; Chief Surgeon to the Widener Memorial Industrial Training School for Crippled Children. The Lenox, Thirteenth and Spruce Sts.

ELECTED

1927. Gilmour, William Rodger, A.B., A.M., M.D., Assistant Surgeon, Philadelphia General and Methodist Hospitals; Instructor in Applied Anatomy, University of Pennsylvania; Associate Professor of Operative Surgery and Surgical Anatomy, Woman's Medical College. 1737 Chestnut St.

1897. Girvin, John H., M.D., Gynecologist to the Presbyterian Hospital; Associate Professor of Gynecology, Graduate School of Medicine, University of Pennsylvania. 2120 Walnut St.

1920. Githens, Thomas Stotesbury, M.D., Chief Pharmacologist, H. K. Mulford Company. Alden Park Manor, Germantown.

1906. Gittings, J. Claxton, M.D., Professor of Pediatrics, School of Medicine and Graduate School of Medicine, University of Pennsylvania; Visiting Physician to the Children's Hospital of Philadelphia. 1828 Pine St.

1905. Given, Ellis E. W., M.D., Surgeon to the Philadelphia Freemasons Memorial Hospital of the Masonic Home, Elizabethtown, Pa.; Surgeon to the Dispensary of the Episcopal Hospital. 2714 Columbia Ave.

1894. Gleason, E. B., S.B., M.D., LL.D., Professor of Otology in the Medico-Chirurgical College. 2112 Pine St.

1906. Goepp, R. Max, M.D., Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. 518 Medical Arts Bldg.

1906. Goldberg, Harold G., M.D., Ophthalmic Surgeon to the Episcopal and Misericordia Hospitals, Kensington Hospital for Women and Baltimore and Ohio Railroad. 2213 Walnut Street.

*1908. Goodman, Edward H., M.D. Dorset, Vt.

1905. Gordon, Alfred, M.D., Neurologist to the Mt. Sinai, the Northwestern General, and the Douglass Memorial Hospitals. 1812 Spruce St.

1894. Graham, Edwin E., M.D., Professor of Pediatrics in the Jefferson Medical College; Pediatrist to the Jefferson and the Philadelphia General Hospitals; Physician to the Franklin Reformatory Home. 1713 Spruce St.

1926. Grant, Francis C., A.B., M.D., Instructor in Surgery, School of Medicine, University of Pennsylvania; Associate in Neuro-Surgery, Graduate School of Medicine, University of Pennsylvania; Visiting Surgeon, Department of Neurological Surgery, Philadelphia General Hospital. 133 S. Thirty-sixth St.

ELECTED

1904. Grayson, Charles P., M.D., Professor of Laryngology and Rhinology in the University of Pennsylvania; Physician-in-Charge of the Throat and Nose Department of the University Hospital; Otolaryngologist to the Philadelphia General Hospital. 262 S. Fifteenth St.

1910. Greenman, Milton J., M.D., Sc.D., Director of the Wistar Institute of Anatomy and Biology. Wistar Institute of Anatomy and Biology, Thirty-sixth St. and Woodland Ave.

1883. Griffith, J. P. Crozer, A.B., M.D., Ph.D., Professor of Pediatrics in the University of Pennsylvania; Corresponding Member of the Société de Pédiatrie de Paris; Physician to the Children's Hospital of Philadelphia. 1810 Spruce St.

1912. Griscom, J. Milton, B.S., M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Attending Surgeon to the Wills Hospital. 2213 Walnut St.

1911. Gummey, Frank Bird, M.D., Visiting Physician to the Germantown Hospital and Dispensary; Visiting Physician to the Midnight Mission. 5418 Greene St., Germantown.

*1902. Gwyn, Norman B., M.D., Clinician in Medicine, Toronto General Hospital; Clinician in Medicine, Medical Faculty, University of Toronto School. 109 Madison Ave., Toronto, Canada.

1894. Hamill, Samuel McC., M.D., Professor of Diseases of Children in the Philadelphia Polyclinic and College for Graduates in Medicine; Pediatrist to the Presbyterian Hospital; Pediatrist to St. Vincent's Home. 1822 Spruce St.

1897. Hand, Alfred, Jr., M.D., Professor of Pediatrics, Graduate School of Medicine, University of Pennsylvania; Visiting Physician to the Children's Hospital, to the Children's Hospital of the Mary J. Drexel Home and to the Methodist Hospital. 1724 Pine St.

1886. Hansell, Howard F., M.D., Emeritus Professor of Ophthalmology in the Jefferson Medical College. N. E. Cor. Seventeenth and Walnut Sts.

1889. Hare, Hobart A., M.D., LL.D., Professor of Therapeutics, Materia Medica, and Diagnosis in the Jefferson Medical College. 1801 Spruce St.

1903. Hart, Charles D., A.M., M.D., Inspector and Secretary of the Eastern State Penitentiary; National Executive Committee and Chairman of the Philadelphia Committee, Boy Scouts of America. Chestnut Hill.

ELECTED

1926. Hartman, Frederick L., A.B., M.D., Clinical Associate in Medicine, Lankenau Hospital; Assistant Instructor in Pathology, University of Pennsylvania; Assistant Pathologist, Visiting Staff of University and Philadelphia General Hospitals. 1914 Pine St.

‡*1888. Hartzell, Milton B., A.M., M.D., LL.D., Professor of Dermatology in the University of Pennsylvania. Fayetteville, Pa.

1907. Hatfield, Charles James, A.B (Princeton), M.D., Executive Director of the Henry Phipps Institute for the Study, Treatment, and Prevention of Tuberculosis; Visiting Physician to the White Haven Sanatorium. Phipps Institute, Seventh and Lombard Sts.

1911. Heed, Charles R., M.D., Ophthalmologist to Girard College; Assistant Ophthalmologist to the Jefferson Medical College Hospital. 1205 Spruce St.

1908. Heineberg, Alfred, P.D., M.D., Associate in Gynecology in the Jefferson Medical College; Assistant Gynecologist to St. Agnes' Hospital. 1923 Spruce St.

1901. Heisler, John C., M.D., Professor of Anatomy, University of Pennsylvania. 3829 Walnut St.

1903. Henry, J. Norman, M.D., Physician to the Pennsylvania Hospital; Clinical Professor of Medicine in the Woman's Medical College of Pennsylvania; Assistant Physician to the Philadelphia General Hospital. 1906 Spruce St.

1927. Herman, Leon, B.S., M.D., Urologist, Methodist Hospital; Assistant Surgeon in Charge of Urology, Pennsylvania Hospital. 1521 Pine St.

1891. Hewson, Addinell, A.B., A.M., M.D., Professor of Anatomy in the Philadelphia Polyclinic and College for Graduates in Medicine; Professor of Anatomy and Histology in the Temple University; Surgeon to Memorial Hospital, Roxborough. 257 S. Sixteenth St.

1924. Hewson, William, A.B., M.D., Assistant in Otology, Graduate School of Medicine, University of Pennsylvania; Otolaryngologist, Pennsylvania Institution for Instruction of Blind and the Shriners' Hospital for Crippled Children; Assistant Otolaryngologist, Memorial Hospital. 6300 Overbrook Ave.

1909. Higbee, William S., M.D., President of the Pennsylvania State Board of Examiners for Registration of Nurses. 5121 Pine St.

ELECTED

1897. Hinkle, William M., M.D., Lecturer on the Anatomy and Physiology of the Vocal Organs in the National School of Elocution and Oratory. 923 N. Sixty-fourth St.

*1892. Hinsdale, Guy, A.M., M.D. Hot Springs, Virginia.

*1888. Hirsh, A. Bern, M.D., Consulting Physician for Physiotherapy, Beth Israel Hospital. 67 W. Ninety-fourth St., New York City.

1888. Hirst, Barton Cooke, A.B., M.D., LL.D., Professor of Obstetrics, Graduate School of Medicine, University of Pennsylvania; Gynecologist to the Philadelphia General and the Howard Hospitals. 1821 Spruce St.

1924. Hirst, John C., 2nd, A.B., M.D., Instructor in Obstetrics, School of Medicine, University of Pennsylvania; Assistant Obstetrican, Philadelphia General and Howard Hospitals; Assistant Surgeon, Department of Obstetrics, University Hospital. 1918 Pine St.

*1908. Hitchens, Arthur Parker, M.D., M.C., U. S. A. Executive Offices, Malacanan, Manila, P.I.

1905. Hodge, Edward Blanchard, A.B., M.D., Surgeon to the Presbyterian and the Germantown Hospitals; Associate Surgeon to the Pennsylvania Hospital; Associate Surgeon to the Widener Memorial Training School. 2019 Spruce St.

‡1913. Hoffman, Clarence, M.D., 1630 Pine St.

1906. Holloway, Thomas B., M.S., M.D., Sc.D. Professor of Ophthalmology, School of Medicine, University of Pennsylvania; Ophthalmologist, Orthopaedic Hospital and in Department of Neurology, Philadelphia General Hospital; Ophthalmologist to the Pennsylvania Institution for the Instruction of the Blind at Overbrook. 1819 Chestnut Street.

1914. Hooker, Richard S., M.D. 2147 N. Howard St.

1919. Hopkins, Arthur H., B.S. (Haverford), M.D. 1726 Pine St.

1925. Hopkinson, R. Dale, M.D., Registrar and Assistant Physician, Abington Memorial Hospital. Jenkintown, Pa.

1908. Hoyt, Daniel M., M.D., Assistant Visiting Physician to the Philadelphia General Hospital. The Tracy, Thirty-sixth and Chestnut Sts.

*1912. Huber, G. Carl, M.D., Professor of Anatomy and Director of the Anatomic Laboratories and Dean of the Graduate School, University of Michigan. 1330 Hill St., Ann Arbor, Mich.

ELECTED

1892. Hughes, William E., M.D., Visiting Physician to the Philadelphia General Hospital; Consulting Physician to the Presbyterian Hospital. 3945 Chestnut St.

1923. Hume, John E., A.B., M.D., Visiting Physician, Medical Department, Howard Hospital. N. E. Cor. Twentieth and Chestnut Sts.

*1912. Hunter, John W., B.S., M.D. McDaniel, Md.

1921. Hunter, Robert J., M.D., Laryngologist, Philadelphia General Hospital; Chief of Dispensary and Assistant Laryngologist and Aurist, Howard Hospital; Instructor in Otology, University of Pennsylvania, Graduate School. 2011 Chestnut St.

1926. Husik, David N., M.D., Associate in Otolaryngology, University of Pennsylvania; Instructor in Otology, Graduate School of Medicine, University of Pennsylvania; Laryngologist and Otologist, Philadelphia General Hospital; Assistant Otolaryngologist, University Hospital. 1610 Spruce St.

1898. Hutchinson, James P., M.D., Surgeon to the Pennsylvania, the Methodist, the Children's, Memorial and the Bryn Mawr Hospitals; Adjunct Professor of Surgery in the University of Pennsylvania. Penn Mutual Life Insurance Company, Sixth and Walnut Sts.

1921. Ivy, Robert H., D.D.S., M.D., Professor of Maxillo-Facial Surgery, Graduate School of Medicine, University of Pennsylvania; Plastic Surgeon to the Hospitals of the Graduate School of Medicine, University of Pennsylvania; Oral Surgeon, Episcopal Hospital; Consultant in Maxillo-Facial Surgery, Walter Reed General Hospital, Washington, D. C. 1503 Medical Arts Building.

1917. Jackson, Chevalier, M.D., Sc.D., Professor of Laryngology, Jefferson Medical College; Professor of Bronchoscopy and Esophagoscopy, Graduate School of Medicine, University of Pennsylvania. 128 S. Tenth St.

*1885. Jackson, Edward, A.M., M.D., Sc.D., Emeritus Professor of Ophthalmology in the University of Colorado; Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic; Secretary of State Commission for the Blind of Colorado. 217 Imperial Bldg., Denver, Colo.

1906. Jacobs, Francis Brinton, B.S., M.D., Pediatrist to the Chester County Hospital. West Chester, Pa.

ELECTED

1922. Jameson, Howard Leon, M.D., Associate Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. 250 S. Seventeenth St.

1920. Janvier, G. Victor, M.D., Assistant Obstetrician University and Philadelphia General Hospitals; Assistant Gynecologist, Philadelphia General and Howard Hospitals. Lansdowne, Pa.

1913. Jefferys, William Hamilton, A.B., A.M., M.D. Rosemont Pa.

1919. Jenks, Horace H., A.B., M.D., Medical Director, Associated Medical Clinics; Physician to the Out-Patient Department, Children's Hospital; Instructor in Pediatrics, Graduate School of Medicine, University of Pennsylvania. 918 Clinton Street.

1926. Johnson, George Ernest, M.D. 5341 Chester Avenue.

1898. Johnson, Russell H., A.B. (Princeton), M.D., Physician to the Pennsylvania Institution for the Deaf and Dumb. Chestnut Hill, Philadelphia.

1918. Jonas, Leon, M.D. 1726 Pine St.

1900. Jones, Charles James, A.M., M.D., LL.D., Ophthalmic Surgeon to St. Joseph's Hospital; Ophthalmic Surgeon to the House of the Good Shepherd, Germantown; Consulting Ophthalmologist to St. Vincent's Home. 626 Medical Arts Bldg.

1924. Jones, Harold W., M.D., Demonstrator of Medicine, Jefferson Medical College; Assistant Physician, Philadelphia General Hospital. 1426 Spruce St.

1913. Jones, John F. X., B.S., A.B., A.M., M.D., Surgeon to St. Joseph's, Misericordia and St. Agnes' Hospitals. 1737 Chestnut St.

1900. Jopson, John H., M.D., Professor of Clinical Surgery, University of Pennsylvania; Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Presbyterian and the Children's Hospitals. 1824 Pine St.

1900. Judson, Charles F., A.B., M.D., Physician to Out-patient Department of the Lankenau Hospital and to the Southern Home for Children. 1005 Spruce St.

1902. Jump, Henry D., M.D., Professor of Applied Therapeutics and Clinical Medicine, Woman's Medical College; Physician to Philadelphia General and Misericordia Hospitals. 2019 Walnut St.

ELECTED

1903. KALTEYER, FREDERICK J., M.D., Associate Professor of Medicine, Jefferson Medical College; Assistant Physician, Jefferson Medical College Hospital; Consulting Physician to the Pottstown Hospital, Pottstown, Pa. 1530 Locust St.

*1910. KARSNER, HOWARD T., M.D., Professor of Pathology in the Western Reserve University Medical School. 2109 Adelbert Rd., Cleveland, Ohio.

1926. KAUFMAN, ISADORE, M.D., Associate in Medicine, University of Pennsylvania; Medical Director, Kensington Dispensary for Tuberculosis; Associate Clinical and Sociological Director, Phipps Institute; Visiting Physician to White Haven Sanatorium. 316 Otis Building.

1923. KAY, JAMES, M.D., Visiting Physician, Episcopal Hospital; Assistant Instructor of Medicine, Jefferson Medical College. 600 W. Olney Ave.

1920. KEATING, PETER MCCALL, M.D., Chief of Surgical Dispensary, St. Agnes Hospital; Assistant Attending Surgeon, Bryn Mawr Hospital; Assistant, Surgical Out-patient Department, Pennsylvania Hospital; Pathologist, Chester County Hospital. Medical Arts Bldg., San Antonio, Texas.

1924. KEELER, J. CLARENCE, M.D., Associate Professor of Otology, Jefferson Medical College and Hospital; Assistant in Oto-laryngology, Germantown Hospital; Consultant Oto-laryngologist, Pottstown Hospital, Pa., and Newcombe Hospital, Vineland, N. J. Medical Arts Building.

†1867. KEEN, WILLIAM W., M.D., LL.D. (Hon.), Ph.D. (Brown, Upsala, etc.) (Hon.), Sc.D. (Jefferson and Harvard), Dr., Honoris Causa, University of Paris; (Hon.) F.R.C.S. (Eng., Edin. and Ireland). 1520 Spruce St.

1912. KEENE, FLOYD E., M.D., Instructor in Gynecology in the University of Pennsylvania; Assistant Gynecologist to the University Hospital; Gynecologist to the Chestnut Hill Hospital. Medical Arts Building.

1913. KELLY, FRANCIS JOSEPH, M.D. 2211 Locust St.

*1887. KELLY, HOWARD A., A.B., M.D., LL.D. (Aberdeen, Wash. and Lee, and Univ. of Pa.), Professor of Gynecology in Johns Hopkins University and Gynecologist to the Johns Hopkins Hospital, Baltimore, Md.; Hon. Fellow of the Edinburgh Obstetrical Society, the Royal Academy of Medicine of Ireland, and of the Glasgow Obstetrical and Gynecological Society. 1418 Eutaw Place, Baltimore, Maryland.

ELECTED

1909. Kelly, James A., A.M., M.D., Visiting Surgeon to St. Mary's and Memorial Hospitals; Associate in Surgery in the Philadelphia Polyclinic and College for Graduates in Medicine; Assisting Visiting Surgeon to St. Joseph's Hospital. 1900 S. Rittenhouse Square.

1912. Kelly, Thomas C., A.M., M.D., Associate in Medicine, University of Pennsylvania; Assistant Professor of Medicine, Graduate School, University of Pennsylvania; Pediatrist to St. Christopher, Misericordia and St. Mary's Hospitals; Assistant Physician, Philadelphia General Hospital. 105 School Lane, Germantown.

1898. Kempton, Augustus F., M.D. 2118 Pine St.

1905. Kercher, Delno E., M.D. 1534 Pine St.

1921. Kern, Richard A., A.B., M.D., Associate in Medicine, University of Pennsylvania; Assistant Physician, University Hospital; Associate in Medicine, Graduate School of Medicine, University of Pennsylvania. 906 Medical Arts Building.

1920. Klauder, Joseph Victor, A.B., M.D., Professor of Dermatology, Woman's Medical College; Associate Professor, Dermatology and Syphilology, Graduate School of Medicine, University of Pennsylvania; Dermatologist, Misericordia, Woman's Medical College and American Stomach Hospitals; Assistant Dermatologist, Philadelphia General Hospital. 1934 Spruce Street.

1920. Klein, Thomas, M.D., Associate Professor of Medicine, Graduate School, University of Pennsylvania; Assistant Physician, Presbyterian Hospital. 250 S. Eighteenth St.

1913. Klopp, Edward J., M.D., Associate in Surgery in the Jefferson Medical College; Assistant Surgeon to the Jefferson Hospital; Surgeon and Chief of the Out-patient Clinic of the Pennsylvania Hospital; Surgeon to the Delaware County Hospital. 1611 Spruce St.

1895. Kneass, Samuel S., M.D., Associate in the William Pepper Laboratory of Clinical Medicine in the University of Pennsylvania. 1510 Walnut St.

1908. Knipe, Jay C., M.D., Ophthalmologist to the Jewish and Mary J. Drexel Hospitals; Associate in Ophthalmology, Jefferson Medical College; Chief of Eye Clinic, Jefferson Hospital. Central Medical Bldg., Eighteenth and Chesnut Sts.

ELECTED

1919. Knipe, Norman Leslie, Ph.B. (Cornell); M.D., Instructor in Obstetrics, University of Pennsylvania; Assistant Obstetrician, University Hospital; Gynecologist, St. Mary's Hospital; Associate Gynecologist, American Stomach Hospital. 2018 Chestnut St.

1908. Knowles, Frank Crozer, M.D., Professor of Dermatology, Jefferson Medical College; Dermatologist to the Presbyterian and Children's Hospitals; Chief of Clinic, Pennsylvania Hospital. 2022 Spruce St.

1925. Knox, Harry E., M.D., Instructor in Surgery, Graduate School of Medicine, University of Pennsylvania; Assistant Surgeon, Germantown, St. Christopher's and Episcopal Hospitals; Surgeon to Philadelphia Hospital for Contagious Diseases. 719 Sixty-sixth Ave., Oak Lane, Phila.

1914. Kolmer, John A., M.D., Dr. P.H., M.Sc., Dr. S.C., LL.D., Professor of Pathology and Bacteriology, Graduate School of Medicine, University of Pennsylvania; Head of the Department of Pathology and Bacteriology Research Institute for Cutaneous Medicine; Consulting Pathologist to St. Vincent's, Misericordia and Memorial Hospitals. Cynwyd, Pa.

1904. Krauss, Frederick, M.D., Ophthalmic Surgeon to the Episcopal Hospital; Ophthalmic Surgeon to St. Christopher's Hospital for Children; Laryngologist to the Abington Hospital. 1701 Chestnut St.

1914. Krumbhaar, Edward B., A.B., Ph.D., M.D., Director of Laboratories, Philadelphia General Hospital; Associate Professor of Pathology, Graduate School of Medicine, University of Pennsylvania. Box 4310, Chestnut Hill.

1900. Krusen, Wilmer, M.D., Professor of Gynecology in the Medical Department of Temple University; Chief Gynecologist to the Samaritan and the Garretson Hospitals; Consulting Gynecologist to the Charity and Mercy Hospitals. 127 N. Twentieth St.

1904. Landis, Henry R. M., M.D., Director of the Clinical and Sociological Departments of the Henry Phipps Institute of the University of Pennsylvania; Visiting Physician to the White Haven Sanatorium. 1817 Pine St.

1907. Langdon, H. Maxwell, M.D., Associate Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Instructor of Ophthalmology, Under-

ELECTED

Graduate School, University of Pennsylvania; Ophthalmologist of the Presbyterian, Orthopædic and Children's Hospitals. 1530 Locust St.

1922. Lawrance, Stuart J., M.D., Obstetrician, St. Mary's Hospital. 1332 Spruce St.

1926. Laws, George M., B.S., M.D., Associate Gynecologist, Presbyterian Hospital; Gynecologist, American Hospital for Diseases of the Stomach. 1907 Spruce St.

1920. Leavitt, Frederick Headley, M.D. 1527 Pine St.

1904. Le Boutillier, Theodore, M.D., Assistant Professor, Epidemic Contagious Diseases, Graduate School of Medicine, University of Pennsylvania; Senior Pediatrist, Philadelphia Hospital for Contagious Diseases; Visiting Physician, Babies' Hospital, North Philadelphia. 2017 Spruce St.

1908. Lee, Walter Estell, M.D., Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Pennsylvania, Germantown, Bryn Mawr and Children's Hospitals, and to the Hospital of the Graduate School of Medicine, University of Pennsylvania. 905 Pine St.

1903. Leffmann, Henry, A.M., M.D., D.D.S., Ph.D., Honorary Professor of Chemistry, Wagner Free Institute of Science; Emeritus Pathological Chemist, Jefferson Hospital; Lecturer on Research, Philadelphia College of Pharmacy and Science. 1729 N. Seventeenth St.

1892. Leidy, Joseph, M.D., Officier d'instruction publique, France; Consulting Physician to the Pennsylvania Training School for Feeble-minded Children. 1713 Walnut St.

1920. Leopold, Simon S., M.D., Associate in Medicine, University of Pennsylvania; Assistant Physician University Hospital. 2025 Spruce St.

1915. Lewis, Fielding O., M.D., Associate in Laryngology in the Jefferson Medical College Hospital; Operating Clinical Chief of the Laryngological Dispensary and Clinical Assistant of the Otological Dispensary of the Jefferson Medical College Hospital; Laryngologist of the Philadelphia General Hospital. 261 S. Seventeenth St.

1877. Lewis, Morris J., M.D., Attending Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases and Emeritus Physician to the Pennsylvania Hospital. Ambler, Pa.

*1911. Lewis, Paul A., M.D. Princeton, N. J.

ELECTED

1904. Lindauer, Eugene, M.D., Assistant Neurologist to the Philadelphia General and Polyclinic Hospitals; Instructor in Nervous Diseases, Graduate School of Medicine, University of Pennsylvania. 6451 N. Broad St.

1927. Lindsey, Walter H., A.B., M.D., Assistant Physician, Episcopal Hospital; Associate in Cardiology, Graduate School of Medicine, University of Pennsylvania. S. E. Cor. Seventeenth and Arch Sts.

1886. Lloyd, J. Hendrie, A.M. (Princeton), M.D., Neurologist to the Methodist Episcopal Hospital; Consulting Neurologist to the State Asylum for the Chronic Insane at Wernersville, and to the Pennsylvania Training School for Feeble-minded Children at Elwyn. 4057 Spruce St.

1907. Lodholz, Edward, M.D., Isaac Ott Professor of Physiology, Graduate School of Medicine, University of Pennsylvania. Medical Laboratories, University of Pennsylvania.

1893. Longaker, Daniel, M.D., Obstetrician to the Kensington Hospital for Women and Visiting Obstetrician to the Jewish Maternity Hospital. 1806 Spruce St.

1907. Loux, Hiram R., M.D., Professor of Genito-urinary Surgery in the Jefferson Medical College; Surgeon to the Philadelphia General Hospital. Medical Arts Building, Sixteenth and Walnut Sts.

1921. Lucké, Baldwin, Dr. P.H., M.D., Assistant Pathologist, University and Philadelphia General Hospitals; Assistant Professor of Pathology, Medical School, University of Pennsylvania.

1924. Ludy, John B., A.B., A.M., M.D., Dermatologist and Chief of Clinic, Pennsylvania Hospital; Dermatologist, Lankenau, Methodist and Delaware County Hospitals. Medical Arts Building.

1923. Lueders, Charles W., A.B., M.D., Biochemist, Pennsylvania Hospital for Mental and Nervous Diseases; Research Worker, Gastro-enterology, Jefferson Medical College. Medical Arts Building.

1919. Lynch, Frank B., Jr., M.D., Dickson Fellow in Bacteriology to the Pepper Laboratory of Clinical Medicine, University of Pennsylvania; Serologist, University Hospital; Instructor in Pathology, Evans Institute, University of Pennsylvania. 6028 Columbia Ave.

1914. Lyon, B. B. Vincent, A.B. (Williams Coll.), M.D. (Johns Hopkins), Associate in Medicine, Jefferson Medical College; Chief-of-Clinic, Gastro-Intestinal Department, Jefferson Hospital. 328 S. Twenty-first St.

ELECTED

1900. McCarthy, Daniel J., M.D., Professor of Medical Jurisprudence (George B. Wood Foundation) in the University of Pennsylvania; Neurologist to the Philadelphia General and St. Agnes' Hospitals, and to the Henry Phipps Institute. 2025 Walnut St.

1913. McCrae, Thomas, B.A., M.D., F.R.C.P. (Lond.), Professor of Medicine in the Jefferson Medical College; Physician to the Jefferson and Pennsylvania Hospitals. 1929 Spruce Street.

1895. McFarland, Joseph, M.D., Sc.D., Professor of Pathology in the Medical Department of the University of Pennsylvania; Pathologist to the Philadelphia General Hospital. 442 W. Stafford St., Germantown.

1905. McKenzie, R. Tait, M.D., LL.D., Professor of Physical Education and Director of the Department of Physical Education in the University of Pennsylvania. 2014 Pine St.

*1916. McKnight, Howard A., A.B., M.D. Tecumseh Bldg., Springfield, Ohio.

1915. McLean, John D., M.D. 135 S. Thirty-sixth St.

1924. McMillan, Thomas M., A.B., M.D., Assistant Physician, Pennsylvania Hospital; Cardiologist, Philadelphia General Hospital; Assistant Instructor in Medicine, School of Medicine, University of Pennsylvania; Instructor in Medicine, Graduate School of Medicine, University of Pennsylvania. 2044 Locust St.

1924. McPhedran, F. Maurice, A.B., M.D., Assistant in Clinical Research, Henry Phipps Institute. Seventh and Lombard Sts.

*1900. McReynolds, Robert Phillips, M.D. 213 S. Broadway, Los Angeles, Calif.

1910. Mackinney, William H., M.D., Professor of Urology, Graduate School of Medicine, University of Pennsylvania; Genito-urinary Surgeon, Philadelphia General Hospital; Chief Urologic Clinic, Lankenau Hospital. 1701 Chestnut St.

1901. MacLeod, George I., M.D. Ardmore, Pa.

1914. Maier, F. Hurst, M.D., Associate in Gynecology to the Jefferson Medical College; Gynecologist to St. Joseph's Hospital; Gynecologist, Obstetrician and Consultant, Prenatal Clinic, Philadelphia General Hospital; Associate Gynecologist, St. Mary's Hospital. 2019 Walnut St.

1913. Major, Charles P., B.S., M.D., Physician to the Dispensary of the Germantown Hospital; Pediatrist to the Dispensary of the Germantown Hospital; Pediatrist to the Abington Memorial Hospital. Tenth and Oak Lane.

ELECTED

1913. Manges, Willis F., M.D., Roentgenologist to the Jefferson Hospital; Director of the Roentgen-ray Laboratory in the Philadelphia General Hospital. 235 S. Fifteenth St.

1898. Marshall, George Morley, M.D., Laryngologist to the Philadelphia General Hospital; Laryngologist and Otologist to St. Joseph's Hospital. 1819 Spruce St.

1889. Martin, Edward, M.D., Professor of Surgical Physiology, University of Pennsylvania. 135 S. Eighteenth St.

*1911. Meigs, Edward Browning, A.B., M.D., Physiologist in the Dairy Division of the United States Department of Agriculture. 1736 M. St., N.W., Washington, D. C.

1914. Mencke, J. Bernhard, A.B., M.D., Assistant Surgeon to the Lankenau Hospital; Surgeon to the Out-patient Department of the Lankenau Hospital. 1816 Spruce St.

1923. Mendelson, Walter, M.D. 639 Church Lane, Germantown.

1914. Merrill, William Jackson, A.B., M.D., Associate Professor of Orthopædic Surgery, Graduate School of Medicine, University of Pennsylvania; Orthopædic Surgeon to the Children's Hospital, Germantown Hospital, Children's Seashore House; Consulting Orthopædic Surgeon to the Howard Hospital and Glen Mills School. 2017 Spruce St.

*1894. Miller, D. J. Milton, M.D., Associate Physician to the Children's Hospital, Philadelphia; Pediatrist to the Bamberger Home for Invalid Children, Longport, N. J. N. W. Cor. Pacific and California Aves., Atlantic City, N. J.

1922. Miller, Edwin B., M.D., Ophthalmologist to Stetson and Northwestern General Hospitals. 2008 Walnut St.

1921. Miller, T. Grier, A.B., M.D., Assistant Physician, University Hospital; Associate in Medicine, University of Pennsylvania. 110 S. Twentieth St.

1881. Mills, Charles K., M.D., LL.D., Emeritus Professor of Neurology in University of Pennsylvania; Neurologist to Philadelphia General Hospital; Consulting Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases. 2121 DeLancey St.

*1917. Mitchell, A. Graeme, M.D., Professor of Pediatrics, College of Medicine, University of Cincinnati; Director of Pediatric and Contagious Services, Cincinnati General Hospital; Chief of Staff of Children's Hospital, Cincinnati. Cincinnati General Hospital.

1904. Mitchell, Charles F., M.D., Surgeon to the Pennsylvania, Bryn Mawr, and Germantown Hospitals. 2003 Pine St.

ELECTED

1921. Mohler, Henry K., P.D., M.D., Associate in Medicine, Jefferson Medical College; Medical Director, Jefferson Hospital. 319 S. Sixteenth St.

*1908. Montgomery, Charles M., A.B., M.D., St. Joseph's Sanatorium, Asheville, N. C.

‡1882. Montgomery, Edward E., A.M., M.D., LL.D., Emeritus Professor of Gynecology, Jefferson Medical College and Gynecologist to St. Joseph's Hospital, Retired. Veree Road, Fox Chase.

1923. Moore, William F., M.D., Assistant, Bronchoscopic Clinic, Jefferson Hospital; Bronchoscopist to Philadelphia General Hospital and Philadelphia Hospital for Contagious Diseases; Assistant Laryngologist, Children's Hospital, Out-patient Department; Laryngologist to Tuberculosis Department, Philadelphia General Hospital. 255 S. Sixteenth St.

1924. Moorhead, Stirling W., A.B., M.D., Associate in Urology, School of Medicine, University of Pennsylvania; Assistant Surgeon, Department of Genito-urinary Surgery and in Urological Dispensary, University Hospital; Urologist, Howard Hospital. 1523 Pine St.

1918. Morgan, Arthur C., M.D., Professor of Applied Therapeutics, Medical Department, Temple University; Visiting Physician to Tuberculosis Department, Philadelphia General Hospital; Consultant to the Eastern State Penitentiary. 2018 Chestnut St.

1886. Morris, Caspar, M.D. 2050 Locust St.

1893. Morris, Elliston J., M.D., Consulting Physician to the Episcopal Hospital. 128 S. Eighteenth St.

1883. Morris, Henry, M.D., Emeritus Professor of Anatomy in the Woman's Medical College of Pennsylvania; Consulting Physician to St. Joseph's Hospital. 2310 Pine St.

1897. Morton, Samuel W., M.D. 2049 Chestnut St.

1905. Müller, George P., M.D., Professor of Clinical Surgery, University of Pennsylvania; Surgeon to the University and Misericordia Hospitals. 1930 Spruce St.

*1915. Musser, John H., Jr., B.S., M.D., Professor of Medicine and Head of Department, School of Medicine, Tulane University of Louisiana. 1551 Canal St., New Orleans, La.

1905. Mutschler, Louis H., M.D., Surgeon to the Episcopal Hospital; Assistant Surgeon to the Orthopædic Hospital. 1625 Spruce St.

*1896. Myers, T. D., M.D. 831 University Ave., Palo Alto, Calif.

ELECTED

1902. Nassau, Charles F., M.D., LL.D., Associate Professor of Surgery in the Jefferson Medical College; Surgeon to St. Joseph's Hospital; Chief Surgeon to the Frankford Hospital; Surgeon to Girard College. 1710 Locust St.

1886. Neff, Joseph S., A.M., M.D., LL.D., D.P.H., Narberth, Pa.

1887. Neilson, Thomas Rundle, A.M., M.D., Surgeon Emeritus to the Episcopal Hospital; Consulting Surgeon to St. Christopher's Hospital for Children; Emeritus Professor of Genito-urinary Surgery in the University of Pennsylvania. 1937 Chestnut St.

1905. Newcomet, William S., M.D., Physician in Charge of Radiological Department, Jefferson, Presbyterian and Oncologic Hospitals. 3501 Baring St.

1905. Newlin, Arthur, B.S., M.D., Physician to the Pennsylvania Hospital; Physician to the Dispensary of the Children's Hospital; Assistant Physician to the Orthopædic Hospital. 1804 Pine St.

1899. Nicholson, William Rufus, A.B., M.D., Professor of Gynecology, Graduate School of Medicine, University of Pennsylvania; Attending Gynecologist and Attending Obstetrician to the Methodist Episcopal Hospital; Attending Obstetrician to the Presbyterian Hospital. 2023 Spruce St.

1889. Noble, Charles P., M.D., Sc.D. (Hon.), Formerly Surgeon-in-Chief, Kensington Hospital for Women; Formerly Clinical Professor of Gynecology, Woman's Medical College. Radnor, Pa.

1905. Norris, Charles C., M.D., Director of the Department of Obstetrics and Gynecology and Professor of Obstetrics and Gynecology, University of Pennsylvania; Gynecologist and Obstetrician University and Philadelphia General Hospitals, Radiological Department, Philadelphia General Hospital and Henry Phipps Institute; Gynecologist to the Children's Hospital, Coronado, Twenty-second and Chestnut Sts.

1905. Norris, George William, A.B., M.D., Professor of Clinical Medicine in the University of Pennsylvania; Physician-in-Chief, Medical Service "A", Pennsylvania Hospital; Attending Specialist (Heart and Lungs), U. S. Public Health Service (Veterans' Bureau). 321 S. Eleventh St.

*1901. Norris, Henry, M.D., Surgeon to the Ruthe ford Hospital. Waverly Mills, S. C.

1892. Norris, Richard C., M.D., Lecturer on Clinical and Operative Obstetrics in the University of Pennsylvania; Obstetrician in Charge of the Preston Retreat; Visiting Obstetrician to the Philadelphia General Hospital; Gynecologist to the

ELECTED

Methodist Episcopal Hospital, and Consulting Obstetrician and Attending Gynecologist to the Southeastern Dispensary and Hospital. 500 N. Twentieth St.

1913. O'Neal, Alexander H., A.B., A.M., M.D., Physician to the Cathcart and Richardson Homes, Devon; Anesthetizer to the Bryn Mawr Hospital. St. Davids, Pa.

1924. Opie, Eugene L., A.B., M.D., Professor of Experimental Pathology, University of Pennsylvania; Director of Laboratories, Henry Phipps Institute. 2203 St. James Place.

1920. Ostheimer, Alfred J., A.B., M.D., L.R.C.P. (London), M.R.C.S. (England), Consulting Neurologist, State Hospital, Norristown, Pa. 6725 Ridge Ave.

1903. Ostheimer, Maurice, A.B., M.D. Whitford, Pa.

1913. Outerbridge, George W., A.B., M.D., Associate Professor of Gynecology, Graduate School of Medicine, University of Pennsylvania; Assistant Gynecologist to the Methodist Hospital; Gynecologist to the Abington Memorial Hospital. 1927 Spruce St.

1915. Owen, Hubley R., M.D., Professor of Clinical Surgery, Woman's Medical College; Surgeon, Philadelphia General Hospital; Assistant Surgeon, Orthopædic Hospital; Chief Surgeon, Bureaus of Police and Fire, Philadelphia. 319 S. Sixteenth St.

1897. Packard, Francis R., M.D., Chief of the Ear, Nose and Throat Service, Pennsylvania Hospital; Consulting Laryngologist and Aurist, Bryn Mawr Hospital; Consulting Aurist Chestnut Hill Hospital; Laryngologist, Children's Hospital of Philadelphia. 304 S. Nineteenth St.

1898. Page, Henry F., M.D., Assistant Physician to the Lankenau Hospital and Physician to the Medical Dispensary of the same; Clinical Professor of Medicine in the Woman's Medical College of Pennsylvania. 315 S. Sixteenth St.

1906. Pancoast, Henry K., M.D., Professor of Roentgenology in the University of Pennsylvania and Roentgenologist to the University Hospital. Ardmore, Pa.

1909. Parish, Benjamin D., B.S., M.D., Associate Professor in Otology, Graduate School of Medicine, University of Pennsylvania; Aurist and Laryngologist to St. Agnes' and Chestnut Hill Hospitals. 1927 Spruce St.

1899. Parke, William E., M.D., Consulting Obstetrician to the Episcopal Hospital; Associate Surgeon to the Kensington

ELECTED

Hospital for Women; Gynecologist to the Frankford Hospital. 1534 N. Fifteenth St.

1925. PATTEN, CLARENCE A., M.D., Assistant Professor of Neurology, Graduate School of Medicine, University of Pennsylvania; Consulting Neurologist Shriner's Hospital for Crippled Children and Delaware County Hospital; Neurologist, Delaware General Hospital. 2037 DeLancey St.

1910. PATTERSON, ROSS VERNET, M.D., Dean and Associate Professor of Medicine, Jefferson Medical College; Physician to the Philadelphia General Hospital; Physician to the Protestant Episcopal Hospital; Assistant Physician to the Jefferson Hospital. 2126 Spruce St.

1926. PAUL, JOHN RODMAN, A.B., M.D., Director of the Ayer Clinical Laboratory; Associate in Surgical Pathology, Graduate School of Medicine, University of Pennsylvania; Fellow in Department of Research Medicine, University of Pennsylvania. 1941 Panama St.

1922. PELOUZE, P. S., M.D., Associate in Urology, University of Pennsylvania; Surgeon-in-Charge, Urological Dispensary, University Hospital. 1737 Chestnut St.

1909. PEMBERTON, RALPH, M.S., M.D., Physician to the Presbyterian Hospital; Director of the Department of Clinical Chemistry in the Pathological Laboratory of the Presbyterian Hospital. 2120 Sansom St.

1914. PEPPER, O. H. PERRY, B.S., M.D., Assistant Professor of Medicine, University of Pennsylvania. Medical Arts Bldg.

†1902. PEPPER, WILLIAM, A.B., M.D., Dean of the School of Medicine, University of Pennsylvania; Member of the Council on Medical Education and Hospitals of the American Medical Association. University of Pennsylvania.

1916. PERCIVAL, MILTON FRASER, M.D. 2332 S. Broad St.

1917. PETER, LUTHER C., A.M., M.D., Professor of Diseases of the Eye, Temple Medical School; Professor of Ophthalmology, University of Pennsylvania Graduate School. N. E. Cor. Twentieth and Chestnut Sts.

1912. PETTY, ORLANDO H., B.S., A.M., M.D., Professor of Diseases of Metabolism, Graduate School of Medicine, University of Pennsylvania; Chief of the Department of Diseases of Metabolism, Hospitals of Graduate School of Medicine, University of Pennsylvania; Chief of Department of Metabolism, Philadelphia General Hospital. 1803 Pine St.

1905. PFAHLER, GEORGE E., M.D., D.O.R.E. (Camb.), Professor of Radiology and Vice-Dean, Department of Radiology,

ELECTED

Graduate School of Medicine, University of Pennsylvania; Director of Radiological Department, Misericordia Hospital. 1321 Spruce St.

1915. Pfeiffer, Damon B., A.B., M.D., Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon, Methodist and Abington Memorial Hospitals; Assistant Surgeon, Presbyterian and Lankenau Hospitals. 1822 Pine St.

1907. Pfromm, George W., Ph.G., M.D., Assistant Physician to the American Stomach Hospital; Consulting Physician to the German Protestant Home for the Aged. 1431 N. Fifteenth St.

1907. Phillips, Horace, M.D., Assistant Surgeon, U. S. Public Health Service; Examining Physician, Office of the District Attorney. 905 Land Title Building.

1911. Piersol, George Morris, B.S., M.D., Professor of Medicine and Vice-Dean for Medicine, Graduate School of Medicine, University of Pennsylvania; Physician to the Philadelphia General and Methodist Episcopal Hospitals. 1913 Spruce Street.

1917. Piper, Edmund Brown, B.S., M.D., Associate in Obstetrics, University of Pennsylvania Medical School; Assistant Professor in Obstetrics, University of Pennsylvania Graduate School; Assistant Obstetrician to the University Hospital; Assistant Obstetrician and Gynecologist to the Philadelphia General Hospital. 1936 Spruce St.

1905. Pitfield, Robert L., M.D., Pathologist to the Germantown Hospital; Bacteriologist to the Chestnut Hill Hospital for Lung Diseases. 5211 Wayne Ave.

1922. Pleasants, Henry, Jr., A.B., M.D., Assistant Visiting Physician, Chester County Hospital, West Chester, Pa.; Assistant in Neurological Clinic, Orthopædic Hospital. West Chester, Pa.

1896. Posey, William Campbell, M.D., Ophthalmic Surgeon to the Howard Hospital; Consulting Ophthalmic Surgeon to the Wills Hospital; Chairman of the Commission on the Conservation of Vision for Pennsylvania. Radnor, Pa.

1899. Potts, Charles S., M.D., Professor of Neurology, Graduate School of Medicine, University of Pennsylvania; Neurologist to the Philadelphia General Hospital; Consulting Neurologist, Lankenau Hospital; Consulting Neurologist, Hospital for the Insane, Atlantic County, New Jersey, 2018 Chestnut St.

ELECTED

*1907. Price, George E., M.D. Suite 620, Paulsen Bldg., Spokane, Wash.

*1920. Quicksall, William E., M.D. E. 216 Fifteenth Ave., North, St. Petersburg, Fla.

1908. Radcliffe, McCluney, A.M. (Lafayette), M.D., LL.D., Consulting Ophthalmologist, Presbyterian Hospital; Attending Surgeon to the Wills Hospital. 1906 Chestnut St.

1913. Randall, Alexander, A.M., M.D., Assistant Professor of Surgery in charge of the Department of Urology, University of Pennsylvania. 1306 Medical Arts Building.

1887. Randall, B. Alex., M.A., M.D., Ph.D., Emeritus Professor of Otology in the University of Pennsylvania; Ear Surgeon to the Children's Hospital; Consulting Aurist to the Pennsylvania Institution for the Deaf and Dumb and to Memorial Hospital, Roxborough. 2320 De Lancey St.

1926. Ravdin, I. S., M.D. 1930 Spruce St.

*1904. Ravenel, Mazyck P., M.D., Professor of Preventive Medicine and Bacteriology and Director of the Public Health Laboratory at the University of Missouri. 203 Hicks Ave., Columbia, Mo.

1925. Reeves, Rufus S., B.S., M.D., Associate in Medicine, Episcopal Hospital. 2227 Spruce St.

1920. Rehfuss, Martin E., M.D. N. E. Cor. 16th and Spruce Sts.

1921. Reiff, E. Paul, A.B., M.D., Member of Medical Staff, Methodist Episcopal Hospital. 5051 Chestnut St.

1919. Reimann, Stanley P., M.D., Director of the Research Institute, Lankenau Hospital; Assistant Professor of Experimental Pathology, Graduate School of Medicine, University of Pennsylvania. 516 Arbutus St., Germantown.

1923. Repplier, Sidney, J., M.D., Medical Director, Curtis Hospital, Curtis Publishing Company. Chestnut Hill, Phila.

1906. Rhein, Robert D., M.D., Chief Physician to the Clinic of the American Hospital for Diseases of the Stomach; Physician to the Philadelphia Home for Incurables; Examining Physician to the White Haven Sanatorium. S. W. Cor. Fifteenth and Pine Sts.

*1910. Rhoads, Samuel, M.D. Altadena, Calif.

1919. Richardson, Russell, A.M., M.D., Director of the Laboratory, Methodist Episcopal Hospital; Pathologist to Abington Memorial Hospital and the Associated Medical Clinics; Instructor in Medicine, University of Pennsylvania. 320 S. Sixteenth St.

ELECTED

1919. Ridpath, Robert F., M.D., Professor of Rhino-laryngology, Temple University; Associate Professor of Laryngology, Graduate School of Medicine, University of Pennsylvania; Laryngologist, Samaritan and Jewish Hospitals. 1737 Chestnut St.

1898. Riesman, David, M.D., Professor of Clinical Medicine, University of Pennsylvania; Visiting Physician to the Philadelphia General and University Hospitals; Consulting Physician to the Jewish and Woman's Hospitals. 1520 Spruce St.

1895. Ring, G. Oram, A.M., M.D., Ophthalmic Surgeon to the Episcopal Hospital; Ophthalmologist to the Widener Memorial Home for Crippled Children; Consulting Ophthalmologist to the American Oncologic Hospital. N. E. Cor. Seventeenth and Walnut Sts.

1899. Roberts, Walter, M.D., Professor of Otology in the Philadelphia Polyclinic; Otologist to the Methodist Episcopal Hospital; Laryngologist to the Philadelphia General Hospital. 1921 Spruce St.

1903. Robertson, William Egbert, M.D., Professor of Theory and Practice of Medicine and of Clinical Medicine in Temple University; Physician to the Episcopal, Samaritan and Garretson Hospitals. 327 S. Seventeenth St.

1903. Robinson, William Duffield, Ph.G., M.D. 2012 Mount Vernon St.

1912. Rodman, John Stewart, M.D., Professor of Surgery, Woman's Medical College; Surgeon-in Chief, Woman's College Hospital; Attending Surgeon, Bryn Mawr Hospital; Assistant Surgeon to the Presbyterian Hospital. 1310 Medical Arts Building.

1909. Rosenberger, Randle C., M.D., Professor of Preventive Medicine and Bacteriology in the Jefferson Medical College. 1001 Walnut St.

*1907. Royer, B. Franklin, M.D. 370 Seventh Ave., New York City.

†1905. Rugh, James Torrance, A.B., M.D., Clinical Professor of Orthopædic Surgery in the Woman's Medical College of Pennsylvania; Orthopædic Surgeon to the Jefferson Medical College; Orthopædic Surgeon to the Methodist and the Philadelphia General Hospitals. 1616 Spruce St.

1897. Sailer, Joseph, Ph.B., M.D., Professor of Clinical Medicine in University of Pennsylvania; Physician to the Philadelphia General, the University, and Presbyterian Hospitals. 250 S. Eighteenth St.

ELECTED

1900. Sajous, Charles E. de M., M.D., LL.D., Sc.D., Officer of the Legion of Honor of France and of the Order of Leopold of Belgium; Professor of Endocrinology, Graduate School of Medicine, University of Pennsylvania; Emeritus Professor of Therapeutics in Temple University Medical School. 2043 Walnut St.

1905. Sartain, Paul J., A.M., M.D. 2006 Walnut St.

1908. Sautter, Albert C., M.D., Assistant in the Dispensary for Diseases of the Eye in the University Hospital; Assistant in the Eye Dispensary of the Lankenau Hospital. 1737 Chestnut St.

1927. Saxon, Gordon J., M.D., 1411 Walnut St.

1906. Saylor, Edwin S., M.D., Chief Ophthalmologist to the Diagnostic Hospital of Philadelphia. 1737 Chestnut St.

1920. Scarlett, Hunter W., M.D. 230 S. Twenty-first St.

1917. Schaeffer, Jacob Parsons, A.M., M.D., Ph.D., Sc.D., Professor of Anatomy and Director of the Daniel Baugh Institute of Anatomy of the Jefferson Medical College. 4634 Spruce St.

1899. Schamberg, Jay F., M.D., Professor of Dermatology and Syphilology, Graduate School of Medicine, University of Pennsylvania; Director, Research Institute of Cutaneous Medicine. 1402 Spruce St.

1917. Schnabel, Truman Gross, A.B. (Lehigh), M.D., Associate in Medicine, University of Pennsylvania; Physician, Philadelphia General and Howard Hospitals, and Medical Dispensary University Hospital; Physician in the Medical Out-patient Department, University Hospital. 1704 Pine St.

1917. Schoff, Charles H., M.D. Media, Pa.

1921. Schumann, Edward Armin, A.B., M.D., Obstetrician and Gynecologist, Philadelphia General Hospital; Gynecologist, Frankford Hospital; Lecturer on Obstetrics, Jefferson Medical College; Consulting Gynecologist, Rush Hospital. 1814 Spruce St.

1887. de Schweinitz, George Edmund, A.M., M.D., LL.D., Professor of Ophthalmology in the University of Pennsylvania; Consulting Ophthalmic Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases, The Philadelphia Polyclinic and School for Graduates in Medicine, and the Philadelphia General Hospital; Ophthalmic Surgeon to the University Hospital. 1705 Walnut St.

ELECTED

*1913. de Schweinitz, George Lord, B.S., M.D., Assistant in the Department of Ophthalmology, St. Luke's Hospital, Bethlehem, Pa. 85 East Broad St., Bethlehem, Pa.

1910. Schwenk, Peter N. K., M.A., M.D., Attending Surgeon to the Eye Department of the Pennsylvania Hospital; Attending Surgeon to the Wills Hospital. 1417 N. Broad Street.

1917. Service, Charles A., M.D. Bala, Pa.

1924. Shallow, Thomas A., M.D., Assistant Surgeon, Jefferson Hospital; Demonstrator of Clinical Surgery, Jefferson Medical College; Surgeon to the Philadelphia General Hospital. 3942 Chestnut St.

1908. Shannon, Charles E. G., A.B., M.D., Instructor in Ophthalmology in the Jefferson Medical College; Assistant in the Ophthalmological Clinic at the Jefferson Medical College Hospital; Ophthalmologist to the Seybert Institution. N. E. Corner Seventeenth and Walnut Sts.

1897. Sharpless, William T., M.D., Physician to the Chester County Hospital. West Chester, Pa.

1906. Shields, William G., M.D., Dermatologist to Germantown Hospital; Chief of Dermatological Clinic and Assistant Physician to the Jewish Hospital. 414 School Lane, Germantown.

*1908. Shoemaker, Harlan, A.B., M.D., Chief Surgeon to Los Angeles County Hospital; Chief of the Surgical Staff of the Methodist Hospital and of the Hospital of the Good Samaritan. 521 Bank of Italy Building, Los Angeles, Calif.

†1893. Shoemaker, Harvey, M.D., Visiting Physician to the Sheltering Arms; Consulting Physician to the Southern Home for Destitute Children; Assistant Physician to the Lankenau Hospital; Physician to the Out-patient Department of the Lankenau Hospital. 1727 Spruce St.

†1896. Shoemaker, William T., M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Clinical Professor of Ophthalmology in the Woman's Medical College of Pennsylvania; Ophthalmic Surgeon to Lankenau, Germantown and Pennsylvania Hospitals. 2017 Locust St.

1900. Shumway, Edward Adams, B.S., M.D., Ophthalmic Surgeon to the Lankenau, Children's and Oncologic Hospitals. 1737 Chestnut St.

1903. Sinclair, John Falconer, M.D., Epidemiologist, Presbyterian Hospital; Physician to the Philadelphia Orphan Asylum and to

ELECTED

the Presbyterian Orphanage; Physician to the Home of the Merciful Saviour for Crippled Children. 4103 Walnut St.

1907. Sinkler, Francis Wharton, A.B., M.D., Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases. 1226 Medical Arts Bldg.

1902. Siter, E. Hollingsworth, M.D., Genito-urinary Surgeon to the Philadelphia General Hospital. 2213 DeLancey St.

1920. Skillern, Ross Hall, M.D., Sc.D., Professor of Laryngology, Graduate School of Medicine, University of Pennsylvania; Laryngologist to Medico-Chirurgical Hospital. 1811 Spruce St.

1905. Smith, S. MacCuen, M.D., Professor of Otology in the Jefferson Medical College; Aurist and Laryngologist to the Germantown Hospital; Aurist to the Jewish Hospital; Consulting Aurist to the Memorial Hospital, Roxborough, and to the Oncologic Hospital. 1429 Spruce St.

1927. Smyth, Calvin M., Jr., M.D. 257 S. Twenty-first St.

1919. Smyth, Henry Field, M.D. Wayne, Pa.

1908. Speese, John, M.D., Associate Professor in Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Children's and Presbyterian Hospitals. 1832 Spruce St.

‡1895. Spellissy, Joseph M., A.M., M.D., Visiting Surgeon to St. Joseph's and to the Methodist Episcopal Hospitals; Assistant Surgeon to the Orthopedic Department of the University Hospital. 317 S. Fifteenth St.

1897. Spiller, William G., M.D., Professor of Neurology in the University of Pennsylvania; Consulting Neurologist to the Philadelphia General Hospital. 4409 Pine St.

1894. Stahl, B. Franklin, B.S., Ph.G., M.D. Consulting Surgeon to St. Agnes' Hospital. Haverford, Pa.

1909. Starbuck, J. Clinton, M.D., Physician to the Orphanage, Orphan Society of Philadelphia at Wallingford and to the Glen Mills School, Glen Mills, Pa. 42 E. Washington St., Media, Pa.

1927. Starr, Isaac, Jr., B.S., M.D., Instructor in Pharmacology, University of Pennsylvania, Acting Physician, University Hospital. Chestnut Hill.

1912. Stauffer, Nathan Pennypacker, D.D.S., M.D., Laryngologist and Otologist to the Presbyterian Hospital; to the Philadelphia General Hospital, Tubercular Department; to the Home for Crippled Children. 1900 Rittenhouse Square.

1910. Stellwagen, Thomas C., Jr., M.D., Chief Clinical Assistant in the Out-patient Surgical Department of the Jefferson Medical College Hospital. 220 S. Sixteenth St.

ELECTED

1895. Stengel, Alfred, M.D., Sc.D., Professor of Medicine in the University of Pennsylvania; Physician to the University Hospital and the Pennsylvania Hospital. 1728 Spruce St.

1901. Stevens, Arthur A., M.D., Professor of Materia Medica, Therapeutics and Clinical Medicine in the Woman's Medical College of Pennsylvania; Lecturer on Physical Diagnosis in the University of Pennsylvania; Physician to the Episcopal and St. Agnes' Hospitals. 314 S. Sixteenth Street.

1914. Stewart, Thomas S., B.S. (Univ. of Penna.), M.D. 1532 Pine St.

1925. Stokes, John H., A.B., M.D., Professor of Dermatology and Syphilology, School of Medicine, University of Pennsylvania; Professor of Dermatology, Graduate School of Medicine, University of Pennsylvania; Attending Dermatologist, University and Philadelphia General Hospitals; Dermatologist, Medico-Chirurgical and Polyclinic Hospitals. 3800 Chestnut St.

1921. Stoner, William H., A.M., Ph.C., Pharm. D., M.D., Associate Professor of Biochemistry and Diseases of Metabolism, Graduate School of Medicine, University of Pennsylvania; Assistant Metabolic Physician, Philadelphia General Hospital. Medical Laboratories, University of Pennsylvania.

1898. Stout, George C., M.D., Consulting Laryngologist and Aurist to the Presbyterian Hospital, the Children's Aid Society and the William Penn Charter School. 2015 Walnut St.

1923. Strecker, Edward, A., A.M., M.D., Professor of Nervous and Mental Diseases, Jefferson Medical College; Clinical Professor of Psychiatry and Mental Hygiene, Yale University Medical School; Neurologist, Pennsylvania Hospital. Medical Director of Department for Mental and Nervous Diseases, Pennsylvania Hospital. 345 S. Nineteenth St.

1923. Stroud, William D., B.S., M.D., Physician to the Pennsylvania Hospital and Chief of Out-Patient and Heart Clinics; Associate in Cardiology, Graduate School of Medicine, University of Pennsylvania; Associate in Medicine, School of Medicine, University of Pennsylvania; Physician in Charge of the Children's Heart Hospital, Wynnefield, Pa. 1011 Clinton St.

1923. Strouse, Frederick M., M.D. 1301 Spruce St.

1884. Stryker, Samuel S., M.D., Consulting Physician to the Presbyterian Hospital. 3833 Walnut St.

ELECTED

1927. Sturgis, Samuel B., M.D., Assistant Instructor in Operative Surgery, University of Pennsylvania. Ardmore, Pa.

*1900. Swan, John M., M.D., Attending Physician, Park Avenue Hospital. 457 Park Avenue, Rochester, New York.

1900. Talley, James Ely, A.B., M.D., Professor of Cardiology, Graduate School of Medicine, University of Pennsylvania; Physician to the Presbyterian and Methodist Episcopal Hospitals. 1811 Spruce St.

1886. Taylor, John Madison, A.B. and A.M. (Princeton), M.D., Professor of Physical Therapeutics and Dietetics, Medical Department Temple University. 1504 Pine St.

1887. Taylor, William J., M.D., Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases, and to St. Agnes' Hospital; Consulting Surgeon to the West Philadelphia Hospital for Women. 1825 Pine St.

1886. Taylor, William L., M.D. 2223 Tioga St.

†1910. Thomas, Benjamin A., A.B., A.M., M.D., Vice-Dean and Professor of Urology, Graduate School of Medicine, University of Pennsylvania; Genito-urinary Surgeon, Presbyterian Hospital; Consulting Genito-urinary Surgeon, Pennsylvania Railroad and to the Prison, Philadelphia. 1900 Spruce St.

1912. Thomas, Frank Wister, A.M., M.D., Visiting Physician to "Buttercup Cottage;" Consulting Physician to the Germantown Hospital. 27 E. Mt. Airy Ave.

1907. Thomas, Thomas Turner, M.D., Associate Professor of Applied Anatomy and Associate in Surgery in the University of Pennsylvania; Surgeon to the Philadelphia General Hospital; Assistant Surgeon to the University Hospital. 1737 Chestnut St.

1896. Thorington, James, A.M., M.D., Consulting Ophthalmic Surgeon to the Presbyterian Hospital; Ophthalmologist to the Ellwyn, Pa., Training School for Feeble-minded Children. 2031 Chestnut St.

1927. Thorington, J. Monroe, B.S., A.M., M.D., Instructor in Ophthalmology, University of Pennsylvania; Assistant in Ophthalmology, Presbyterian Hospital. 2031 Chestnut St.

1898. Thornton, Edward Q., M.D., Assistant Professor of Materia Medica in the Jefferson Medical College. 1331 Pine St.

1912. Torrey, Robert G., M.D., Physician to the Philadelphia General Hospital; Associate in Medicine, University of Pennsylvania. 1716 Locust St.

ELECTED

1896. Toulmin, Harry, M.D., Vice-President, Medical Department, Penn Mutual Life Insurance Company. Haverford, Pa.

1908. Tracy, Stephen E., M.D., Gynecologist to the Stetson Hospital; Visiting Surgeon to the Gynecean Hospital. 1737 Chestnut St.

1926. Tucker, Gabriel, M.D., Associate Professor of Bronchoscopy and Esophagoscopy, Graduate School of Medicine and Associate Bronchoscopic Surgeon, University Hospital, University of Pennsylvania; Associate in Bronchoscopy and Assistant Bronchoscopist, Jefferson Medical College and Hospital; Bronchoscopist, Misericordia and Bryn Mawr Hospitals. Cynwyd, Pa.

*1901. Tucker, Henry, M.D. Bellevue, Md.

†1894. Tunis, Joseph Price, M.D., Assistant Professor of Histology and Embryology, Temple University. Chestnut Hill, Philadelphia.

1924. Turner, Charles Root, A.B., D.D.S., M.D., Dean, School of Dentistry, University of Pennsylvania. Merion, Pa.

1923. Turner, Creighton H., M.D., Associate in Medicine, Jefferson Medical College; Visiting Physician, St. Agnes' Hospital. 1731 Pine St.

1897. Tyson, T. Mellor, M.D., Physician to the Philadelphia General Hospital; Physician to the Rush Hospital, the Philadelphia Lying-in-Charity Hospital and the Children's Aid Society of Philadelphia. 1506 Spruce St.

1907. Ullom, Josephus Tucker, M.A., M.D., Physician to the Chestnut Hill Hospital. Carpenter and Quincy Streets, Germantown.

*1913. Vail, William Penn, B.S., M.S., M.D., Laryngologist to the Pennsylvania Institution for the Instruction of the Blind; Laryngologist to the Department for Tuberculosis of the Philadelphia General Hospital; Assistant Laryngologist and Otologist to the Out-patient Department of the Pennsylvania Hospital; Assistant Laryngologist to the Children's Hospital. Blairstown, N. J.

°1873. Van Harlingen, Arthur, Ph.B. (Yale), M.D., Emeritus Professor of Diseases of the Skin in the Philadelphia Polyclinic. Bryn Mawr, Pa.

1903. Van Pelt, William Turner, M.D. Chestnut Hill, Philadelphia.

ELECTED

1893. VANSANT, EUGENE LARUE, M.D. 1737 Chestnut St.

1912. VAUX, NORRIS WISTAR, M.D., Surgeon to the Chestnut Hill Hospital; Surgeon to the Dispensary of the Germantown Hospital. 1810 S. Rittenhouse Square.

*1897. VEASEY, CLARENCE A., A.M., M.D. Suite 404, Paulsen Building, Spokane, Wash.

†1883. VINTON, CHARLES HARROD, A.M., M.D. P. O. Box 464, Atlantic City, N. J.

1903. WADSWORTH, WILLIAM SCOTT, M.D. 3914 Baltimore Ave.

1927. WAGERS, ARTHUR J., M.D., Assistant Professor of Laryngology Graduate School of Medicine; University of Pennsylvania; Demonstrator of Otology and Assistant Demonstrator of Laryngology, Jefferson Medical College; Otologist, Joseph Price Hospital. The Lenox, Thirteenth and Spruce Sts.

1906. WALKER, JOHN K., M.D., Physician to the Children's Hospital of the Mary J. Drexel Home; Physician to the Out-patient Department of the Pennsylvania Hospital. 1814 Pine St.

1907. WALKER, WARREN, M.D., Dermatologist to the Episcopal and Bryn Mawr Hospitals. 1819 Chestnut St.

1904. WALSH, JOSEPH, A.M., M.D., Visiting Physician to and Medical Director of the White Haven Sanatorium. Medical Arts Building.

‡1910. WARD, E. TILLSON, A.M., M.D. 2006 Mt. Vernon St.

1895. WATSON, ARTHUR W., M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic and College for Graduates in Medicine; Laryngologist to the Jewish Hospital; Laryngologist and Aurist to the Mt. Sinai Hospital; Laryngologist to Home for Incurables. 2013 Locust St.

1923. WATSON, WILLIAM R., M.D., Chief Laryngologist, Episcopal and Diagnostic Hospitals; Assistant Chief Laryngologist, Pennsylvania Hospital. 2124 Pine St.

1903. WEBER, CHARLES H., M.D., Associate in Pediatrics, Graduate School of Medicine, University of Pennsylvania; Physician to the Medical Dispensary, Children's Hospital; Pediatrist, Howard Hospital and to the Municipal Court. 2048 Pine St.

1920. WEIDMAN, FRED. D., M.D., Professor of Dermatological Research, University of Pennsylvania; Assistant Pathologist, Philadelphia Zoölogical Garden; Attending Dermatologist, Philadelphia General Hospital. 20 Tenby Road, Llanerch, Pa.

ELECTED

1906. Weisenburg, Theodore H., M.D., Professor of Neuro-Psychiatry, Graduate School of Medicine, University of Pennsylvania; Neurologist, Philadelphia General Hospital and Physician to Orthopædic Hospital and Infirmary for Nervous Diseases. 2037 DeLancey St.

1923. Weiss, Edward, M.D., Physician to the Tuberculosis Wards, Philadelphia General Hospital; Associate in Pathology, Jefferson Medical College. 1923 Spruce St.

1913. Wells, P. Frailey, A.M., M.D., Ph.D., 754 N. Fortieth St.

1893. Westcott, Thompson S., M.D., Pediatrist to the Methodist Episcopal Hospital; Consulting Physician, Haddock Memorial Home. 1720 Pine St.

1921. Whitaker, William, M.D., Surgeon to Dispensary of the Episcopal Hospital. 5448 Germantown Ave.

1901. White, Courtland Y., M.D., Director of the Pathological Laboratories of the Episcopal Hospital; Pathologist to the Children's Hospital and to the Kensington Hospital for Women; Chief Bacteriologist to the Bureau of Health, Department of Health and Charities, Philadelphia. 6611 N. Tenth St.

*1905. Whiteway, Harold M., M.D. 2356 W. Twenty-first St., Los Angeles, Calif.

1898. Whiting, Albert D., M.D., Associate Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Associate Surgeon, Lankenau Hospital; Surgeon, Germantown Hospital. 333 S. Eighteenth St.

1925. Widmann, Bernard P., M.D., 1321 Spruce St.

1914. Willard, de Forest P., B.S. (Univ. of Penna.), M.D., Professor of Orthopædics, Graduate School of Medicine, University of Pennsylvania; Orthopædic Surgeon to the Polyclinic and Delaware Hospitals and Shriners' Hospital for Crippled Children. 1729 Spruce St.

1907. Williams, Carl, B.S., M.D., Ophthalmic Surgeon to the Germantown Hospital; Instructor in Ophthalmology in the University of Pennsylvania; Ophthalmic Surgeon to the Pennsylvania Institution for the Deaf and Dumb. School Lane and Greene Sts., Germantown.

1924. Williams, Horace James, M.D. 5908 Greene St. Germantown.

1920. Williams, Philip F., Ph.B., M.D., Assistant Professor of Obstetrics, Graduate School of Medicine, University of Pennsylvania; Instructor in Obstetrics, School of Medicine, University of Pennsylvania; Obstetrician to the Maternity Hospital of Philadelphia and Assistant Gynecologist to the Presbyterian Hospital. 2206 Locust St.

ELECTED

1927. Williamson, Ernest G., M.D., Surgeon, Children's Hospital; Assistant House Surgeon, Pennsylvania Hospital; Assistant Surgeon, Presbyterian and Jefferson (Dispensary) Hospitals. 6353 Woodbine Ave.

1916. Wilmer, Harry B., M.D., Assistant Instructor in Medicine, University of Pennsylvania; Assistant Visiting Physician and Assistant Neurologist to the Germantown Hospital; Visiting Chief to the Dispensary of the Germantown Hospital. 138 W. Walnut Lane, Germantown.

1921. Wilson, George, M.D., Assistant Professor of Neurology, School of Medicine, University of Pennsylvania; Clinical Professor of Neurology, Women's Medical College of Pennsylvania; Neurologist, Philadelphia General, Episcopal and Abington Memorial Hospitals; Assistant Neurologist, University Hospital. 2025 Walnut St.

1874. Wilson, James Cornelius, A.M. (Princeton), M.D., Emeritus Professor of the Practice of Medicine and of Clinical Medicine in the Jefferson Medical College; Physician-in-Chief (Emeritus) to the Lankenau Hospital; Emeritus Physician to the Pennsylvania Hospital; Consulting Physician to the Bryn Mawr Hospital. 1509 Walnut St.

1902. Wilson, Samuel M., M.D. 2018 Chestnut St.

1897. Wilson, W. Reynolds, M.D. Villa Nova, Pa.

1904. Wister, James W., M.D., Physician to the Out-patient Department of the Germantown Hospital. 5430 Germantown Ave.

*1901. Witmer, A. Ferree, M.D. 21 North Grand Ave., Baldwin, N. Y.

1918. Wolferth, Charles Christian, A.B., M.D., Associate in Medicine, University of Pennsylvania; Associate in Cardiac Pathology, William Pepper Laboratory of Clinical Medicine, University of Pennsylvania. 1704 Pine St.

1893. Wood, Alfred C., M.D., Assistant Professor of Surgery in the University of Pennsylvania; Surgeon to the University, the Philadelphia General, St. Timothy's and the Howard Hospitals. 2035 Walnut St.

1900. Wood, George, B., M.D., Laryngologist and Otologist, Orthopædic and Howard Hospitals; Professor of Laryngology, Graduate School, University of Pennsylvania. N. E. Cor. Twentieth and Chestnut Sts.

1903. Wood, Horatio C., Jr., M.D., Professor of Pharmacology and Therapeutics in the University of Pennsylvania; Pro-

ELECTED

fessor of Materia Medica, Philadelphia College of Pharmacy and Science. 319 S. Forty-first St.

‡°1880. WOODBURY, FRANK, M.D., Secretary to the Committee on Lunacy of the Board of Charities of Pennsylvania. 101 Allen Lane, Mt. Airy.

*1911. WOODS, ANDREW H., A.B., M.D., Professor and Head of the Neurological Department, Peking Union Medical College, Peking, China.

†1897. WOODWARD, GEORGE, M.D. W. Willow Grove Ave., Chestnut Hill, Philadelphia.

1903. WORDEN, CHARLES B., A.M., M.D. Princeton, N. J.

1894. ZENTMAYER, WILLIAM, M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Attending Surgeon to the Wills Hospital; Ophthalmologist to the Glen Mills School. 1506 Spruce St.

1887. ZIEGLER, WALTER M. L., A.M., M.D. 1418 N. Seventeenth St.

1895. ZIMMERMAN, MASON W., M.D., Consulting Ophthalmic Surgeon to the Germantown Hospital. 1518 Waverly St.

ASSOCIATE FELLOWS

(Limited to Fifty, of whom Twenty may be Foreigners)

American

ELECTED

1911. Abbe, Robert, M.D. 40 W. Fifty-ninth St., New York City, New York.

1909. Billings, Frank, M.D., 1550 North State Parkway, Chicago, Illinois.

1880. Carrow, Flemming, M.D., 823 David Whitney Building, Detroit, Mich.

1893. Councilman, William T., M.D., Harvard Medical College, Boston, Massachusetts.

1909. Crile, George W., M.D., 1021 Prospect Avenue, S. E., Cleveland, Ohio.

1909. Dana, Charles Loomis, M.D., 53 West Fifty-third Street, New York City, New York.

1892. Emmet, Thomas Addis, M.D., 91 Madison Avenue, New York City, New York.

1916. Estes, William Lawrence, M.D., South Bethlehem, Pa.

1912. McCaw, Brig.-Gen. Walter D., M.D. Assistant Surgeon-General, U. S. A., Army Medical School, Washington, D. C.

1906. Mayo, William J., M.D., Rochester, Minnesota.

1917. Miner, Charles H., M.D., Wilkes-Barre, Pa.

1906. Pilcher, Lewis Stephen, M.D., 145 Gates Avenue, Brooklyn, New York.

1906. Shattuck, Frederick C., M.D., 135 Marlborough Street, Boston, Massachusetts.

‡1894. Warren, J. Collins, M.D., 58 Beacon Street, Boston, Massachusetts.

‡1894. Weir, Robert F., M.D., 16 East Ninety-sixth St., New York City, New York.

1892. Welch, William H., M.D., Johns Hopkins Hospital, Baltimore, Maryland.

Foreign

1924. Hopkins, Sir F. Gowland, M.D., Cambridge, England.
1909. Macallum, Archibald B., M.A., M.B., Ph.D., Sc.D., LL.D., F.R.S., Department of Biochemistry, McGill University, Montreal, Canada.
1922. McCarrison, Lieut.-Col., Robert, M.D., C.I.E., D.Sc. LL.D., F.R.C.P. (London), Pasteur Institute, Coonoor, S. India.
1924. Macleod, John James R., M.D., M.B., D.Sc. (Hon.), LL.D., F.R.S. 43 Nanton Ave., Toronto, Canada.
1906. Myles, Sir Thomas, M.D., 33 Merion Square, W., Dublin, Ireland.
1885. Rendu, Jean, M.D., Lyons, France.
1908. Ross, Col. Sir Ronald, K.C.B., K.C.M.G., F.R.S., M.D., D.Sc., LL.D., Ross Institute and Hospital for Tropical Diseases, Putney Heath, London, S.W. 15, England.

NECROLOGICAL LIST

FELLOWS

MILTON B. HARTZELL, M.D.	April	7, 1927
E. E. MONTGOMERY, M.D.	April	17, 1927
E. TILLSON WARD, M.D.	April	20, 1927
JOHN G. CLARK, M.D.	May	4, 1927
FRANK WOODBURY, M.D.	May	24, 1927
JOSEPH M. SPELLISSY, M.D.	July	27, 1927
CLARENCE HOFFMAN, M.D.	September	21, 1927
J. COLLINS WARREN, M.D.	November	3, 1927
GUIDO BACCELLI, M.D.	January	10, 1916

LIST OF PRIZES AND LECTURES

THE WILLIAM F. JENKS MEMORIAL PRIZE

(Triennial)

1889	John Strahan, M.D.	Belfast (Ireland).
1895	Abram Brothers, M.D.	New York.

Note—June 12, 1900, the William F. Jenks Memorial Prize Fund was transferred to the Library as the "William F. Jenks Memorial Library Fund."

ALVARENGA PRIZE OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA

(Annual)

1890	R. W. Philip, M.D.	Edinburgh (Scotland).
1891	L. Duncan Bulkley, M.D.	New York.
1892	R. H. L. Bibb, M.D.	Saltillo.
1894	G. E. de Schweinitz, M.D.	Philadelphia.
1895	Guy Hinsdale, M.D.	Philadelphia.
1897	Joseph Collins, M.D.	New York.
1898	S. A. Knopf, M.D.	New York.
1898	Robert Randolph, M.D.	Baltimore.
1900	David de Beck, M.D.	Cincinnati.
1901	George W. Crile, M.D.	Cleveland.
1903	William S. Carter, M.D.	Galveston.
1905	D. Chalmers Watson, M.D.	Edinburgh (Scotland)
1907	William Louis Chapman, M.D.	Providence.
1908	William T. Shoemaker, M.D.	Philadelphia.
1910	M. Katzenstein, M.D.	Berlin (Germany).
1911	Francis D. Patterson, M.D.	Philadelphia.
1914	H. B. Sheffield, M.D.	New York.
1915	J. E. Sweet, M.D.	Philadelphia.
1917	Wilburt C. Davison, M.D.	Baltimore.
1921	John W. Churchman, M.D.	New York.
1923	Edward P. Heller, M.D.	Kansas City, Mo.
1924	Gordon Cameron, M.B., B.S.	Victoria, Australia.
1925	Raphael Isaacs, M.D.	Boston.
1926	P. S. Pelouze, M.D., and Frederick S. Schofield, M.D.	Philadelphia.
1927	Emil Bogen, M.D.	Cincinnati, Ohio.

NATHAN LEWIS HATFIELD PRIZE FOR ORIGINAL RESEARCH IN MEDICINE

(Triennial)

1901	Henry F. Harris, M.D.	Atlanta.
1909	Martin Henry Fischer, M.D.	Oakland.
1917	A. B. Macallum, M.D., F.R.S.	Toronto.
1919	Harvey Cushing, M.D.	Boston.
1921	William R. Nicholson, M.D.	Philadelphia.
1922	Sir Thomas Lewis, C.B.E., M.D., F.R.S.	London (Eng.).
1923	Frederick Grant Banting, M.D.,	Toronto.
1924	George J. Heuer, M.D.	Cincinnati.
1926	Knud Faber, M.D.	Copenhagen.

NOTE.—November 29, 1913, by Supplemental Deed of Trust, the title of this Fund was changed to "Nathan Lewis Hatfield Prize and Lectureship."

WEIR MITCHELL LECTURES

Jan. 17, 1911	Arthur R. Cushny, M.D.	London.
Mar. 30, 1911	Edmund B. Wilson, Ph.D., LL.D.	New York.
May 16, 1911	Svante Arrhenius	Stockholm.
Nov. 3, 1911	William T. Porter, M.D.	Boston.
Mar. 29, 1912	William H. Howell, M.D.	Baltimore.
Oct. 21, 1912	G. H. F. Nuttall, F.R.S., M.D.	Cambridge (Eng.).
April 4, 1913	H. P. Armsby, Ph.D., LL.D.	Pennsylvania.
Feb. 25, 1914	Harvey Cushing, M.D.	Boston.

PUBLIC LECTURES

Feb. 16, 1910	S. Weir Mitchell, M.D.	Philadelphia.
Nov. 17, 1910	Simon Flexner, M.D.	New York.
Dec. 15, 1910	William H. Welch, M.D.	Baltimore.
April 18, 1911	James G. Mumford, M.D.	Clifton Springs, N.Y.
Nov. 20, 1911	Talcott Williams, A.M., LL.D., Litt.D.	Philadelphia.
April 29, 1912	Owen Wister, A.M., LL.D.	Philadelphia.
Feb. 17, 1913	John K. Mitchell, M.D.	Philadelphia.
Feb. 15, 1916	Daniel J. McCarthy, M.D. and Walter Estell Lee, M.D.	Philadelphia.
April 15, 1916	Surgeon A. M. Fauntleroy	U. S. Navy

MARY SCOTT NEWBOLD LECTURES

Mar. 29, 1917	A. T. McCormack, M.D.	Bowling Green, Ky.
April 4, 1919	Col. Thomas W. Salmon, M.C.,	U. S. Army.
Feb. 6, 1920	Alonzo E. Taylor, M.D.	Philadelphia.
Feb. 4, 1921	Maj.-Gen. Merritte W. Ireland	Washington.
April 20, 1921	Prof. William Romaine Newbold,	Philadelphia.
Nov. 11, 1921	Robert McCarrison, M.D., D.Sc., LL.D.	Oxford (Eng.).
Feb. 1, 1922	Hans Zinsser, M.D.	New York.
April 5, 1922	G. Canby Robinson, M.D.	Baltimore.
Dec. 6, 1922	Donald C. Balfour, M.D.	Rochester, Minn.
Mar. 7, 1923	J. J. R. Maeleod, M.D.	Toronto.
April 4, 1923	Prof. Leon Asher, M.D.	Berne (Switzerl'd).
Mar. 5, 1924	Prof. A Biedl	Prague (Czecho-Slovakia).
April 15, 1924	J. G. FitzGerald, M.D.	Toronto.
Oct. 31, 1924	John I. Hunter, M.D., and N. D. Royle, M.D.	Sydney (Australia).
Dec. 4, 1924	Willem Einthoven, M.D.	Leyden (Holland).
April 21, 1925	A. N. Richards, A.M., Ph.D.	Philadelphia.
Feb. 2, 1927	Eugene L. Opie, M.D.	Philadelphia.
Nov. 2, 1927	George R. Minot	Boston.

MÜTTER LECTURES

1865	J. H. Packard, M.D.	Philadelphia.
1866	J. H. Packard, M.D.	Philadelphia.
1867	J. H. Packard, M.D.	Philadelphia.
1868	Harrison Allen, M.D.	Philadelphia.
1869	J. H. Brinton, M.D.	Philadelphia.
1872	J. da S. Solis Cohen, M.D.	Philadelphia.
1879	S. W. Gross, M.D.	Philadelphia.
1882	E. O. Shakespeare, M.D.	Philadelphia.
1885	H. F. Formad, M.D.	Philadelphia.
1888	O. H. Allis, M.D.	Philadelphia.
1890–1891	Roswell Park, M.D.	Buffalo.
1893–1894	De Forrest Willard, M.D., and Guy Hinsdale, M.D.	Philadelphia.
1896	O. H. Allis, M.D.	Philadelphia.
1899–1900	J. B. Roberts, M.D.	Philadelphia.
1901	H. W. Cushing, M. D.	Boston.
1902	L. A. La Garde, M.D.	Washington.
1903	C. N. B. Camac, M.D.	New York.

1904	G. H. Monks, M.D.	Boston.
1905	A. O. J. Kelly, M.D.	Philadelphia.
1906	W. J. Mayo, M.D.	Rochester, Minn.
1907	J. Rogers, M.D., and S. P. Beebe, M.D.	New York.
1908	G. W. Crile, M.D.	Cleveland.
1909	H. D. Fry, M.D,	Washington.
1910	T. W. Hastings, M.D.	New York.
1911	C. F. Nassau, M.D.	Philadelphia.
1912	J. C. Bloodgood, M.D.	Baltimore.
1913	R. C. Coffey, M.D.	Portland, Ore.
1914	F. H. Albee, M.D.	New York.
1915–1916	Rudolph Matas, M.D.	New Orleans, La.
1916	Nelson M. Percy, M.D.	Chicago, Ill.
1917	Chevalier Jackson, M.D.	Philadelphia.
1919	Alexis V. Moschcowitz, M.D.	New York.
1920	J. Chalmers Da Costa, M.D.	Philadelphia.
1921	Prof. H. C. Jacobaeus	Stockholm (Swed.).
1922	James Ewing, M.D.	New York.
1923	J. Edwin Sweet, M.D.	Philadelphia.
1924	Evarts Graham, M.D.	St. Louis.
1925	Edward Archibald, M.D.	Montreal, Can.
1926	Walton Martin, M.D.	Ithaca, N. Y.
1927	E. Starr Judd, M.D.	Rochester, Minn.

WEIR MITCHELL ORATION

1919	Charles W. Burr, M.D.	Philadelphia.
1922	Charles Loomis Dana, M.D	New York.
1925	Simon Flexner, M.D.	New York.

MEMOIR OF HENRY REDWOOD WHARTON, A.M., M.D.*

By JOHN H. JOPSON, M.D.

Dr. Henry Redwood Wharton was born in Philadelphia, May 23, 1852. On his father's side he was descended from Thomas Wharton, an English Quaker gentleman, who came to Philadelphia from Westmorelandshire the year after Penn arrived, and who in 1688 married Rachel Thomas, of Monmouthshire, Wales. From this pair are descended the many Whartons who in this country have distinguished themselves in public life and in private enterprise. Sturdy merchants, learned lawyers, able legislators and statesmen, literary lights, and, in spite of their Quaker blood, brave and skilful soldiers are thick on the family tree. Medicine claimed a few of the name, but more who were related through the female side of their families. The history of this virile and prolific stock has been admirably portrayed by that talented historian and authority on Colonial history, Miss Anne Hollingsworth Wharton, the sister of the subject of this sketch. He was also descended through the female side from the Carpenters and the Hollingsworths, and the blood of these famous Quaker families mingled in his veins with that of Thomas Wharton and his Welsh wife. It is of interest to recall that old Joseph Wharton, or Duke Wharton, as he was generally known, was Dr. Wharton's great-great-grandfather. It was at his country mansion in 1778, two years after his death, that the famous Meschianza was held, as described in Weir Mitchell's novel, *Hugh Wynne*. Joseph Wharton had eighteen children, one of whom was Robert Wharton, that famous mayor who served thirteen terms and did many notable things in and out of office.

Dr. Wharton's mother was Mary McLanahan, of Greencastle, Pa., and of Scotch-Irish extraction. Her father, Dr. John Boggs, was a widely-known physician in that section, sometime surgeon in the War of 1812, and the son of Major Boggs of the Continental Army.

* Read January 12, 1927.

Charles Wharton, Dr. Wharton's father, was an ironmaster and owned a furnace at Chestnut Grove, between Gettysburg and Carlisle. He had a country house there, where the family spent their summers and here Henry Wharton passed his vacations as a boy, hunting and fishing, and imbibing that taste for field sports which was one of his distinguishing characteristics in later life. He was there as a boy of eleven, with his aunt, Miss Isabel Boggs, afterward Mrs. de Schweinitz, during the battle of Gettysburg, and by his quick wit prevented a party of Confederate raiders from finding and confiscating his father's horses. The winters were usually spent in Philadelphia, where he attended school, first at the Friends' Central School under the tutelage of the well-remembered Aaron Ivins, and later at the F. W. Smith classical school, where he prepared for college. His education was continued at the University of Pennsylvania, where he was a member of the Class of 1873, along with Walter George Smith, John W. Brock, Henry Pleasants and William B. Abbey. Graduating with the degree of Bachelor of Arts, he studied medicine at the same institution, his class, that of 1876, being the first to graduate under the new three-year requirement, and the second to graduate after removal of the Medical Department to the new site in West Philadelphia. Agnew and John Neill were the professors of surgery, to which subject young Wharton's tastes already inclined, as shown by the fact that while still a student he successfully trephined his favorite hound, who had suffered a depressed fracture of the skull. The patient was restored to full earning capacity in the hunting field, although partially deaf. Dr. Wharton's graduation thesis was on "Foreign Bodies in the Brain."

The University Hospital had been opened in 1874, and, on graduating, Wharton received an appointment as interne. Here he came under the immediate influence of Agnew, while in the following year John Ashhurst, Jr., was elected professor of clinical surgery as successor to Neill. A youngster was indeed fortunate in having the opportunity of serving under both these strong men. Agnew was in his prime, brilliant operator, skilled anatomist, and favorite practitioner. Ashhurst typified the scholar in surgery, already recognized as one of the great literary lights of his profession, a fine diagnostician, and one who had operated boldly and written extensively and well on lesions of the joints, the bloodvessels, and the brain.

Dr. Richard H. Harte was a student in the Medical Department

at that time and followed Dr. Wharton as interne in the Hospital. Many were the anecdotes they loved to tell of those happy days. There was an old and experienced orderly in the Hospital, such a character as Treves could have painted in an essay like the one he wrote on "The Old Receiving Room." After Ashhurst's first clinic, for which they were all keyed up and which was a long and heavy one, the orderly exclaimed, "My God, the Professor will kill us all!" Hunter was there assisting Agnew, but doomed to an early end of a promising career. Deaver was just graduating, while J. William White was beginning his career, with the ambition, industry and forceful determination which were to land him in the John Rhea Barton chair. The new surgery was not yet born in this country, and surgeons still wore their old bloodstained cloth coats, and sometimes whetted their scalpels on their boots in preparation for the amputation of a limb or a breast, the excision of a tumor or a joint, the ligation of a bloodvessel, or the operation of cutting for stone. But the surgical type was much the same then as now, and, as Fred Lund once said, first in classical language and then in English, "There were heroes before Agamemnon" and "There were good surgeons before Charlie Mayo was born."

Following the University Hospital service, and a further term as interne at the Germantown Hospital, Wharton entered practice in Philadelphia. He declined a tempting offer to go with Dr. Dunton, of Germantown, as an assistant, when the practice of that suburb was in the hands of the Three Ds—Dunton, Darragh and Downs. But Ashhurst had already invited him to become his assistant at the University, which offer he accepted, opened an office in his father's house, and began his career as hospital surgeon and practitioner. The duties of assistant surgeon to the University Hospital on the Ashhurst service were shared with his close friend, Harte. Naturally one of the most industrious of men, to the University Hospital appointment was quickly added that of visiting surgeon to the old Children's Hospital, where the Ashhursts, John and Samuel, were the other surgeons, and later to the staff of the Mary J. Drexel Hospital and the Pennsylvania Institute for the Deaf and Dumb. He taught and quizzed at the University, and de Schweinitz and Hare were among his pupils. He was active in the various medical societies and wrote a number of papers during this period, especially on subjects dealing with pediatric surgery, in which field his experience was unusually large. He became the best-known tracheot-

omist and intubator in the city at a period when antitoxins were unheard of and when diphtheria levelled a heavy toll among the children of rich and poor alike. In 1891, when well established in practice, he was elected surgeon to the Presbyterian Hospital, and from then until his retirement it was with this hospital and with the Children's Hospital that he was most intimately identified. Connected for a time as visiting surgeon at the Methodist Hospital, and on the original staff at Bryn Mawr, he early resigned from the former, but continued as consulting surgeon at the latter until his death. He was consulting surgeon to the St. Christopher's Hospital. He was also for many years consulting surgeon to the Girard College, the duties of which position were not confined to lending his name, but entailed active attendance on the sick and injured boys domiciled in that admirable institution, and where his broad experience in pediatric surgery was peculiarly useful. There he had an almost, if not quite, unbroken record of recoveries from operations for acute appendicitis, which were sufficiently common. I remember well his observations on the much greater danger attending acute osteomyelitis in youths under the same conditions, which he noted in his service there.

At the Presbyterian Hospital his position was quite unique. He was president of the staff for fourteen years. He never missed his daily visit, except during his brief vacations, usually scattered through the hunting season. Here he referred practically all his private patients who required hospitalization. He had an important practice in this city, and he was on terms of personal friendship with many of his patients, who loved him greatly. He had the respect and confidence of all of the staff and the affection of many of them. He was very generally known both to the senior and the junior staff as "Uncle Harry." His popularity with the nursing staff was long and unbroken. He was the favorite surgeon to whom our nursing sisters, to use a good English term, turned for advice and treatment. His simplicity, kindness, mother wit and keen common sense were outstanding features which appealed to men and women alike. To his patients he was surgeon, confident and friend, while his professional confrères knew "Harry" Wharton as one to whom they could turn as a man turns to his friend in time of trouble.

Dr. Wharton continued as demonstrator of surgery at the University until 1900. He delivered the didactic lectures on surgery for two years during the final illness of John Ashhurst. Resigning in

1901, after Ashhurst died, he was elected professor of clinical surgery at the Women's Medical College, which position he held for several years. He served a number of terms on the Council of this College, and was a president of the Academy of Surgery. He was a fellow of the American Surgical Association, which connection he valued very highly, and an original member of the Philadelphia Pediatric Society.

Wharton's contributions to the literature included a number of papers of importance, most of which were contributed to the proceedings of the College and the Academy of Surgery. They included such subjects as "Tracheotomy in Diphtheritic Croup," "The Treatment of Empyema in Children," "Wounds of the Venous Sinuses of the Brain," "Dislocation of the Ulnar Nerve," papers on rectal surgery, and a number dealing with fractures both in children and adults. He was greatly interested in the subject of fractures and very proficient in their treatment. He collaborated as senior author with the late B. Farquahar Curtis, of New York, in writing a *Practice of Surgery,* which appeared in 1897, had three editions, and was an excellent work, ambitious and comprehensive in scope and characterized by sound surgical teaching. The chapter on "Diseases and Injuries of the Eye" was written by his close friend, Dr. de Schweinitz. His *Text-book of Minor Surgery,* the best-known book in its field, ran through nine editions, the last appearing in 1922, and at a time when his health was failing and he had been forced to curtail his hospital work. This last revision was a congenial task to which his physical and mental powers were still equal when more strenuous occupations were denied. He was a contributor to Ashhurst's *Encyclopedia of Surgery,* and to Starr's *American Text-book of the Diseases of Children.* His writings were marked by careful preparation, good judgment, a broad experience and common sense.

Dr. Wharton was an excellent diagnostician and a skilled therapeutist. He was a conscientious, deliberate and painstaking operator, and extremely careful and assiduous in the after-treatment of his operative cases. He excelled in the treatment of injuries and diseases of the bones and joints. He was notably successful in the operations for harelip and cleft-palate. Mention has been made of his skill as tracheotomist and intubator. In the general field of pediatric surgery he was, I believe, excelled by none.

I have alluded to his personal characteristics. The most unassuming and yet the most human of men, he delighted in the society of

his friends and in the personal contacts with his patients. He was a popular member of the Philadelphia and the University clubs, and of the Medical Dining Club. He was fond of outdoor life and of gardening and was the keenest of sportsmen. With him fishing and shooting were lifelong passions. Many were the happy days I shared with him in the field and on the water. He was a good shot, and during the open season was always studying the weather and looking forward to a trip to the Eagle Island Gun Club. Here, like a sensible man, he laid aside for a brief period the cares of practice, and busied himself with the avocations dear to a sportsman's heart.

Dr. Wharton married Miss Edith R. Booth, of New Castle, Delaware, who with five children survives him. Of his four boys, three were of the military age during the World War, and along with his son-in-law served with credit as commissioned officers in the armed forces of the United States. He took a warranted pride in their records. He himself was desirous of active service, but had to content himself with a very useful position as a member of the Medical Advisory Board of the Selective Draft, sitting at the Presbyterian Hospital. The absence of many of the members of the surgical staff of the Hospital, called away by war service, threw a great additional strain on those remaining. To this demand Dr. Wharton, just recovered from a serious illness, rose with his oldtime industry and resourcefulness, working hard and materially increasing the debt which that institution and the community owed him for his self-sacrificing labors. He retired as visiting surgeon and was made consulting surgeon in 1921. For several years before his death he was in failing health, with a series of focal infections, associated with profound anemia. He died in Philadelphia on December 3, 1925.

MEMOIR OF HENRY WEIGHTMAN STELWAGON, M.D.*

By FRANK CROZER KNOWLES, M.D.

Henry Weightman Stelwagon, the son of Joseph and Margaretta (Duckett) Stelwagon, was born in Philadelphia, December 3, 1853. His early education was received in the public schools of his native city and in the Salem Academy of New Jersey. Andalusia College was later entered, from which institution he was graduated, with the degree of B.S. in 1872. Studies were then started in the medical school of the University of Pennsylvania and graduation occurred in 1875. The degree of Doctor of Philosophy was also received from the latter institution. The Philadelphia General Hospital was selected for his resident physician training.

The field of cutaneous medicine having been selected for his life work, study abroad was deemed imperative and the dermatologic clinics of Vienna and Berlin were selected. A more favorable time could hardly have been selected, as Hebra was still actively teaching and Kaposi was in the zenith of his career. Two years were spent under the tutelage of these great masters and a return was then made to Philadelphia.

After his return from postgraduate studies, the first appointment was in charge of the dermatologic department of the Northern Dispensary, with which institution he maintained his consulting affiliation until his death. During the ten-year period 1880–1890 he was in charge of the Philadelphia Dispensary for Skin Diseases. He became instructor in dermatology and chief of the Dispensary for Skin Diseases of the Hospital of the University of Pennsylvania in 1885. These positions were held until 1890. The bulk of the dermatologic instruction during this period fell on his shoulders, due to the poor health of Professor Duhring.

In 1888 he occupied the chair of clinical professor of dermatology in the Woman's Medical College of Pennsylvania, and teaching was continued in this institution until 1907. During the former year he was made dermatologist to the Philadelphia General Hospital

* Read January 10, 1927.

and maintained his active connections with this institution until 1915, later becoming consultant.

There was a long service as dermatologist to the Howard Hospital, running from 1885 to 1912, with subsequently a consultant capacity.

The position at his Alma Mater was resigned in 1890 and the lectureship in dermatology at the Jefferson Medical College and dermatologist to the Jefferson Hospital were accepted. The title of this new chair at Jefferson was shortly changed to clinical professor and later to professor. Teaching in this institution was continued until June, 1918.

Many valuable contributions were made to dermatological literature. In association with Professor Duhring, a chapter was prepared, "Diseases of the Skin," for Pepper's *System of Medicine;* contributions were also made to Wilson's *Applied Therapeutics,* to Keating's *Cyclopedia of Diseases of Children,* to Morrow's *System of Dermatology,* to Hare's *System of Therapeutics,* to Buck's *Handbook of the Medical Sciences* and to Sajous' *Annual and Cyclopedia of Practical Medicine.*

In 1890 a need was realized for a small volume especially for students, and he published his *Essentials of Diseases of the Skin.* Mracek's *Atlas of Skin Diseases* was translated, edited, several new pictures added and the test augmented. In 1901 his *Treatise on Diseases of the Skin* was published and had run through eight editions and several reprintings in eighteen years. There has probably never been a textbook on dermatology which has had such a wide distribution, has been so universally used for teaching and has been so often consulted and quoted by not only the specialist but by those in general practice.

Membership was maintained in the Philadelphia County Medical Society, College of Physicians of Philadelphia, Philadelphia Dermatological Society, Pennsylvania State Medical Society, Historical Society of Philadelphia, American Dermatological Association and the American Medical Association.

Various honors were conferred on him, due to his distinguished position in his chosen field. He was selected as president of the American Dermatological Association in 1900. The chairmanship of the Section of Cutaneous Medicine and Surgery was conferred in 1901. Because of his scientific papers and that remarkable volume on *Diseases of the Skin* several foreign dermatological societies conferred honorary or associate membership: He was an

honorary member of the Society of Dermatology and Syphilology of Italy, associate member of the Society of Dermatology and Syphilology of France, of the Vienna Dermatological Society and of the Berlin Dermatological Society.

During the last few years of his life his health had been somewhat broken and he suffered with an occasional attack of angina pectoris. He was found dead in his office chair, in the late afternoon of October 18, 1919. In the harness until the end, still devoting himself to humanity and to that field of medicine that he loved the most.

Dr. Stelwagon was always immaculately groomed, delightful to everyone, courtesy to the extreme and liked by all.

May I end this all-too-brief memoir with a personable touch. On my desk one of my most prized possessions is a photograph of my former chief, which always brings back memories of thoughtfulness, consideration and friendship.

MEMOIR OF ALLEN J. SMITH, M.D.*

By EDWARD MARTIN, M.D.

Allen J. Smith, A.M., M.D., Sc.D.

Born, 1863.

Gettysburg College, A.B., 1883; A.M., 1886; Sc.D., 1910.

University of Pennsylvania, M.D., 1886.

Magill University, LL.D., 1911.

Assistant Demonstrator of Pathology, University of Pennsylvania, 1887–1891.

Professor of Pathology, University of Texas, 1891–1903 (Dean of Faculty for ten years).

Professor of Pathology and Comparative Pathology and Director of Courses in Tropical Medicine, 1911 (Dean of Medical Faculty, 1909–1912; Acting Dean, 1917–1918), University of Pennsylvania.

Lieutenant-Colonel, Medical Department, U. S. Reserve Corps (active service, June, 1918, to April, 1919).

Died, 1926.

Class Book on Bacteriology. Translations of Kitt's *Comparative General Pathology* and von Fürth's *Problems of Physiological and Pathological Chemistry of Metabolism.*

Medical News prize. Anatomic prize.

Sections in various textbooks upon legal medicine in various modes of death; animal parasites and the principles of immunity; journal articles upon a variety of subjects in pathologic anatomy, parasitology and tropical affections.

Discovery of Bacillus cœruleus (one year after graduation from medical school).

Establishment of hookworm disease as endemic in the United States.

Production of evidence implicating bedbugs as conveyors of leprosy infection and of ticks as harboring larvæ of certain filarial worms.

Incrimination of the mouth ameba as a contributing cause of pyorrhea alveolaris and suggesting emetin as a helpful treatment.

* Read January 10, 1927.

In 1895, Allen J. Smith first observed, and recognized as such, hookworm ova recovered from the feces of the common closet of the Medical Department of the University of Texas. Search for the individual host or hosts was unavailing.

In 1901 he found similar ova in the stools of a patient in St. Mary's infirmary, and, following the administration of thymol and salts recovered more than 100 parasites from the dejecta. The ova from this patient were demonstrated to the medical students who, working over some presumably normal fecal matter taken from the common closet of the school, found similar ova in large numbers. Search for the hosts was again fruitless.

A month later the finding of a distinct eosinophilia in the blood of 2 students exhibiting malarial symptoms suggested an examination of the stools, from which were recovered in both cases hookworm ova. Shortly thereafter Dr. Smith examined the feces of 86 students, coming, in the main from widely different parts of the state of Texas. Eight exhibited hookworm infection.

Dr. Smith's work upon hookworm disease was fundamental and conclusive of the proposition that this affection was endemic in this country. He later realized the portent of his observations and lived to see the inestimable benefits accruing therefrom to our southern states and to many tropical and subtropical lands.

Called in 1891 to the Chair of Pathology, Microscopy and Bacteriology at the University of Texas, just taken over by the state, Dr. Smith found this institution representing little more than an earnest effort toward medical education. He left it in 1903, a school of high standing, to accept the Professorship of Pathology in the University of Pennsylvania.

In Texas he was his own janitor until a slight increase in his budget enabled him to secure at a low price a temperamental leper, who served well for some years until his physical infirmity was recognized by an overbright student and reported to the daily press. As for the leper's mental obliquity, distrained of his liberty at St. Mary's Hospital, he flatly refused that autopsy, the hope for which may have been a contributing cause of his janitorial appointment.

In Texas Dr. Smith taught histology, embryology, inorganic chemistry and lectured on mental and nervous diseases. At his request his title was changed to Professor of Pathology.

Dr. Randall writes that, during his residence in Texas, there came

to Dr. Smith a call to the pathologic professorship of one of the largest and strongest medical schools in this country; with the assurance of an adequate budget, abundant medical and surgical material and a salary, a thousand dollars more than the one he was receiving.

The students called a mass meeting, raised a thousand dollars and presented this to Dr. Smith, with an earnest petition that he stay with them. Both check and the call of the larger institution were refused.

As Dean of the School, ably abetted by such men as Kislen, Thompson and Randall, he inspired that confidence, respect and personal regard which insured both individual and legislative financial support.

He held that the first duty of a teaching department is to teach and carried this doctrine to its ultimate conclusion, using for this purpose the rich autopsy and operative material afforded by hospitals of more than 2000 beds. He particularly stressed the relation borne by pathologic anatomy to disturbances of function and the symptoms resulting therefrom. He and his class habitually became so enthralled by his and their study and description of abnormal findings in a given organ, and symptoms which must have resulted therefrom, that, forgetful of other subjects and professors, they would continue their discussion long past the appointed hour. Disarmingly contrite when protests were made, when he again met his class he and it, each stimulating the other, became amnesic to aught but pathology.

He rejected, and with contumely, the offer of one whose hour followed his, to supply an alarm clock which, harshly ringing at the appointed time, should rapidly release a mephitic vapor.

Even his final examination was a teaching one.

A big, study, large-framed, broad-shouldered, powerful man, pale rather than red, clear steady kindly blue eyes, broad intellectual forehead, the shadow of a smile about the mouth, in part concealed by a short moustache, he sat behind a desk covered with specimens on zinc trays, and animal parasites in jars. To the nervous student a few words, bearing upon his family perhaps, for Dr. Smith had an extraordinarily wide acquaintance, and an even more extraordinary and detailed memory thereof. When human relations had sufficiently softened the official ones, the student was given an organ, for instance, asked to describe his findings and the

effects of such upon its bearer during life. Whatever the student's showing, Dr. Smith ended the examination by a symptomatic reconstruction, chronologically and logically sequential, much as the paleontologist reconstructs from a lower jaw the configuration, size, age and habits of an extinct animal.

Even as Welch, of Hopkins, Smith, of the University of Texas and of the University of Pennsylvania, was one to whom students instinctively turned—for answer to medical queries, solutions of their personal difficulties, advice as to their future or present activities. To him before all others came graduates visiting their Alma Mater, assured of a cordial greeting, a vivid interest in their careers, a clear memory of their undergraduate lives and sympathetic understanding of their problems. None left him without freshened faith in themselves, renewed belief in human kindliness and increased respect and affection for one so learned, so wide, so trustworthy and so elated by their triumphs, so helpful in their trials.

Having definitely crystallized his views upon a given subject, they were not readily subject to the lysis of conflicting opinion, even though this were vigorously expressed by the majority of his faculty colleagues; nor, when overruled did he entertain other feeling than pity for the ninety and nine who were out of step. The struggle over, he was not more cheerful in victory than he was in defeat.

Associated with the encyclopedic brain was an artistic temperament, evidenced by unusual skill as a draftsman and a colorist.

In his college days Allen Smith, in addition to leading his class, from both the popular and scholastic standpoint, devoted his late night and early morning hours to the study of music, majoring in horns. He might have become a tuba virtuoso but for a protesting citizenship, a revolting student body and an unsympathetic faculty.

Into the archeology of Central America he made an excursion to which he frequently referred and with the vividness characteristic of open eyes, wide knowledge and an unusual gift for clear expression.

In the realm of sport he was an occasional duck hunter, taking pleasure therein rather for the companionship of congenial fellow huntsmen than for the kill.

Dr. Smith was an individual worker with a genius for persistence and detail. Among his many publications his *Synopsis of Studies in Metazoan Parasitology*, beautifully illustrated by himself, should

take high rank. Therein are pictured and set forth many parasites heretofore undescribed.

Because of his wide knowledge, large experience and unending patience, he was sought, not only for the identification of parasites, but for the microscopic study of neoplasms, his final judgment on which was accepted without question.

He came into the world a giver, asking no return. In the service of others his strength of body, his clarity of mind, his wealth of knowledge and his material resources were equally available for the asking. He conferred a favor with the air of one who gratefully and gracefully receives it.

His last words were true to type. Past midnight, in the terminal stage of a cardiorenal death, gasping for breath, when Mrs. Smith was calling a doctor, he said, "No, I must fight this out myself."

And shortly thereafter came his lasting rest.

MEMOIR OF JOHN B. ROBERTS, M.D.[1]

By WILLIAM E. ROBERTSON, M.D.

I ESTEEM it a privilege to serve as the mouth-piece of my honored colleagues in voicing our sense of appreciation of one whose life was ineffably plain and wholesome.

In every community there are some whose comings and goings are perennially thrust before the public eye. Possessing no outstanding merits, their utter lack of self-consciousness makes them immune to criticism. Such men are apt to be less concerned with ethical and moral values than they are in attaining their own selfish ends. The antithesis of this type furnishes another group of men—men modest in their make-up, sincere, wholly lacking the spectacular, their diffidence sometimes being mistaken for austerity. Such differences in type must exist, however, as has been pointed out by Herbert Spencer in his book on education.

I have always felt that Dr. Roberts occupied a borderline position. He could afford to be courteous in defeat and uniformly tolerant, for all he ever did in his many activities was not done with the thought of personal aggrandizement, but for the benefit of the common weal, as it was given him to see it.

Lord Macaulay has expressed this thought in discussing the corporate existence of the two great parties which developed in 1641 during the tenure of the Long Parliament. According to Macaulay, "It has its origin in diversities of temper, of understanding and of interest, which are found in all societies, and which will be found until the human mind ceases to be drawn in opposite directions by the charm of habit and by the charm of novelty. Not only in politics, but in literature, in art, in science, in surgery and mechanics, in navigation and agriculture; nay, even in mathematics, we find this distinction. Everywhere there is a class of men who cling with fondness to whatever is ancient, and who, even when convinced by overpowering reasons that innovation would be beneficial, consent to it with many misgivings and forebodings. We find also every-

[1] Read January 10, 1927.

where another class of men, sanguine in hope, bold in speculation, always pressing forward, quick to discern the imperfections of whatever exists, disposed to think lightly of the risks and inconveniences which attend improvements, and disposed to give every change credit for being an improvement. In the sentiments of both classes there is something to approve. But of both, the best specimens will be found not far from the common frontier. The extreme section of one class consists of bigoted dotards; the extreme section of the other consists of shallow and reckless empirics."

No one who knew Dr. Roberts ever failed to appreciate and respect the motive which actuated him in his political activities. Often distasteful, as it must have been for a man of his dignity and quiet tastes, to be brought into intimate association with the common type of professional politician, he graciously accepted it in the concept which he held of his duty as a citizen. Willingly giving both time and money, he refused to accept any emolument. Such fees as are paid to those who serve at the polls, he utilized in his efforts to serve justice and righteousness.

His life attests his concurrence in the teaching of Confucius, that one should not permit his dignity to become supercilious. Possessing a just regard for tradition, he never permitted it to dominate him.

He must have recognized the adumbrations of the postwar period, for he regarded one's birth and social position as merely accidental, and accepted and valued an individual solely upon his merits. Honor and candor were the qualities he admired. He never believed that ambition should stultify these. Even when he realized that he might jeopardize his own position by his frankness, he voiced his convictions with absolute fearlessness. The truth, as he saw it, was paramount. Such men are rare.

Dr. John Ashhurst was such a man. I well remember asking the support of Dr. Ashhurst in my candidacy for a certain position. He said to me, "Do you know there are several other candidates?" I answered, "Yes, sir." He said, "Do you know Dr. So and So?" mentioning the name of one of them. Again I answered, "Yes, sir." He asked me, "Do you think you are as well qualified as he is for the position?" I said, "No, Sir." "Neither do I," he replied. I thanked him for his frankness and left him with a sense of profound respect, almost amounting to reverence.

Those among you who recall some of Dr. Ashhurst's critical reviews may remember that he could be caustic on occasion. How-

ever, his conclusions were based upon a careful reading of the subject; he was always truthful, brief and concise, and appended his initials to whatever he wrote.

Dr. John Ashhurst and Dr. John Roberts had much in common. Both were Christian gentlemen, straightforward almost to bluntness in their candor. Both approached their work in the spirit of profound conscientiousness. Never spectacular, they both planned cautiously and executed deliberately. Both lived through a period, insensible in its march, but none the less epoch-making and wonderful in retrospect. Extremely conservative, both lived to enjoy the benefits conferred by this cataclysmic period of medical history.

Dr. John Bingham Roberts was born February 29, 1852, at 141 North Eleventh Street; then, I believe, No. 39. He was the son of Caleb Cresson and Helen S. Bingham Roberts. He attended the Lawton School in his earlier years, and in 1867, at fifteen years of age he entered the Academic Department of the University of Pennsylvania, graduating third in his class in June, 1871. In September of that year, he entered Jefferson Medical College under the preceptor ship of his uncle, Dr. Richard J. Levis. He graduated in March, 1874, having won the Gross surgical prize, and in the same year received his Master's degree at University of Pennsylvania. Shortly after, he went to Europe, spending part of the years 1874 and 1875 in observation and study.

From October, 1875, to April, 1877, he served as resident physician at the Pennsylvania Hospital, where Dr. Richard J. Levis was one of the visiting surgeons. During this internship he was elected attending physician, and to lecture on clinical medicine at Jefferson Hospital, then recently erected. He resigned in 1878 to devote his entire time to surgery, and in that year again spent some time in study in Europe. He had also been physician to the outpatient department of the Pennsylvania Hospital, but resigned in 1878.

In the spring of 1877 he was elected and served for three years as instructor in surgery in the Jefferson Medical College. In 1878 he revived the Philadelphia School of Anatomy, where he taught anatomy and operative surgery for several years. During part of this time he also served as demonstrator of anatomy in the Philadelphia Dental College.

In 1879 he was invited to assist in organizing the Philadelphia Academy of Surgery and became its first recorder. In 1881 he was appointed to deliver the first annual surgical address. He resigned,

however, with the request that Dr. S. D. Gross be invited to deliver the first oration.

In 1881 he declined the professorship of anatomy in the Medico-Chirurgical College of Philadelphia, and declined the same chair in the Women's Medical College of Pennsylvania and in the College of Physicians and Surgeons of Chicago, likewise in the Chicago Medical College in 1882, and in 1885 in the University of Vermont.

In 1887 he refused to become surgeon to and lecturer upon clinical surgery at Jefferson Medical College, because he did not approve of graduating students after attendance on only two courses of lectures.

In 1882 and 1883 he was the most active spirit in organizing the Philadelphia Polyclinic and College for Graduates in Medicine. At that time the youngest member of the corporators and faculty, he continued to serve this institution until it became the Graduate School of the University of Pennsylvania, in 1918, and for a period of two years thereafter.

In May, 1888, Dr. Roberts graduated from the Medical School of the University of Pennsylvania, and was later appointed lecturer on anatomy in that institution, serving until 1891.

In 1886 he married his cousin, Anna H. Roberts, who died in September of that year. In 1897 he married Mary Eleanor Roberts, the youngest sister of his first wife, who, with two sons and a daughter, survive him.

Dr. Roberts was attending surgeon to St. Mary's Hospital from 1883 to 1889, and became surgeon to the Jewish Hospital in 1887; to St. Agnes Hospital in 1888 to 1889.

From 1889 to 1899 he was professor of the principles and practice of surgery in the Women's Medical College of Pennsylvania, and lecturer on clinical surgery at the Women's Hospital of Philadelphia.

From 1890 to 1899 consulting surgeon to the West Philadelphia Hospital for Women. In 1890 he declined the position of surgeon to the German Hospital on the ground that he was unable to give the necessary time. This is worthy of comment, for it is the only occasion in an unusually busy life that he ever made such a plea.

In 1891 he became visiting surgeon to the Methodist Episcopal Hospital, and served as consulting surgeon to the Kensington Hospital for Women from 1891 to 1899.

His affiliation with the Polyclinic and College for Graduates in Medicine extended from its inception in 1882 up to 1920, two years after its merger with the University of Pennsylvania.

His activity with his pen was equally remarkable. From the time of his first contribution, in 1879, on *Paracentesis of the Pericardium*, a good-sized monograph, almost to the end of his life, he wrote assiduously, as over twenty volumes on the shelves of our library will attest. He revised and enlarged Bryant's *Surgery*, was one of the editors of the American edition of Holmes' *System of Surgery;* published several editions of a *Compend on Anatomy;* a number of books on plastic surgery, including deformities of the face and nose and cleft palate; also several works on fractures; a *Manual of Modern Surgery;* a work entitled, *Field and Limitation of Operative Surgery of the Human Brain;* contributed articles to Ashhurst's *Encyclopedia of Surgery;* Dennis' *System of Surgery;* Pepper's *System of Medicine* and Keen's *Surgery*.

He published many articles in magazines, including such topics as "Modern Medicine and Homeopathy;" papers on ethics; "The Doctor's Duty to the State," and a goodly array of articles on the doctor's responsibility in a civic and political sense.

Few physicians have ever given so freely of their time to medical organizations as Dr. Roberts gave, both in an executive and professional capacity, and still fewer have so willingly aided and given counsel to younger men. His latch-string was always out and he never seemed too busy to give of his time. Courteous, sympathetic, kindly, thoroughly altruistic, he was a gentlemen of the old school, a type which, I fear, is becoming rare.

In medicine, as in politics, he was a reformer. Whatever faults he possessed were the result of his ardent zeal. Conscientious and high-minded in the extreme, an idealist, unwilling, even if able, to see the sordid side of the average human, he often suffered defeat at the hands of those whom he strove to assist. He accepted it with unusual grace, however, and was ever ready to try again, for he always built on faith.

I cannot recount all of his activities in medical societies. Suffice it to say that he had twice been president of the Philadelphia County Medical Society; president of the Pennsylvania State Medical Society; member of the Judicial Council and of the Bureau of Legislation of the American Medical Association; delegate representing that body at the International Medical Congress at London. He addressed the British Medical Association at their meeting in Edinburgh, the title of his paper having been, "Fractures of the Elbow."

For a number of years he was a member of the finance committee of the College of Physicians, and for five years treasurer of this honorable body; several times vice-president and president of the Mutual Aid Society; president of the section on anatomy of the first Pan-American Medical Congress; an original member of the Société internationale de chirurgie; president of the Philadelphia Academy of Surgery; vice-president of the Association of the American Medical Colleges; president of the American Academy of Medicine. A fellow of the American Surgical Association since 1882, he has been vice-president in 1888; in 1920 he was elected president, an honor which he valued as highly as any he had ever received. His failing vision made it impossible to assume the chair, however.

He was nominated twice for Common Councils and once for the State Legislature, but failed of election. This very failure, particularly in the murky state of politics then existing, was an unintentional tribute to the sterling qualities of Dr. Roberts as a man.

Dr. Roberts was a man of untiring energy and industry, to whom, as we have seen, came many well-merited honors. When we consider the fact that for years he struggled with poor vision, having myopia, his accomplishments are more remarkable. In his later years he suffered repeated attacks of retinal hemorrhage, and, though almost blind, retained a keen mind, an interest in many things and a cheerful disposition. Never a word of complaint or allusion to his infirmity, he served as a lesson to many younger men unhandicapped, for his interest in medicine and medical societies continued unabated up to the day of his injury.

In the morning of October 7, 1924, cheerful and kindly as usual, with his mind filled with plans devised for the help and benefit of his fellows, his infirmity made a peculiar impress upon me. I trembled for his safety, and asked permission to accompany him on a mission he felt it his duty to perform. Kindly, but firmly, he refused the proffered help. A few minutes later he was struck by a truck in crossing Broad and Walnut Streets. He sustained a severe scalp wound and cerebral concussion from which he never recovered.

For years he had been a trustee of Holy Trinity Episcopal Church, and I believe his life attests the fact that he endeavored to carry out in a practical way his religious convictions.

He died November 28, 1924.

ANNUAL ADDRESS OF THE PRESIDENT*

By HOBART A. HARE, M.D.

A PRESIDENT of the College of Physicians who has already made an address on three previous occasions naturally finds little new for the consideration of the Fellows as he is about to take leave of his office under the rules of the College. There are several points, however, which were touched upon in my last address that may be considered once more.

Each department of the College, without doubt, needs more funds and gifts from those who are Fellows, or from others, and gifts designed for the benefit of special funds are earnestly desired. In the past these needs have been frequently recognized. Nevertheless, it would be very advantageous if the general fund of the College for maintenance could be increased. This fund might be given, or left by bequest, not to any specific committee or department, but to the College itself in the way of endowment, and from this source the College, or its Council, could from time to time appropriate money for special purposes. Furthermore, if the objection be raised that the giving of such a fund, or funds, would have no recognition in the sense that the donor's name would not be attached, this difficulty could be met by dividing up the general maintenance funds into special entities, to which the donor's name could be attached. When the authorities of the College saw fit to make an appropriation for a given purpose it could be announced that the Hall Committee, for example, had been aided by the Thomas Smith Fund to the extent of —— dollars, or that the Library Committee had been enabled to purchase, or care, for certain valuable books through the J. Fund.

While it can be truthfully said that no committee or part of the College is at present receiving what it needs, there is always danger of the main institution being neglected, whereas its departments may be well provided for. Thus a bequest to a given committee might consist of a piece of property which at the time of gift was not

* Read January 9, 1928.

particularly valuable, yet which if left intact might greatly increase in value. Perhaps no better illustration of this could be cited than the endowment of the chair of anatomy in the University of Edinburgh. This sum was relatively small at the time of the bequest, but in later years brought to the holder of that chair a salary far in excess of any ever paid any teacher anywhere. If it had been given to the medical school instead of to a single chair its proper distribution would have done far more good, instead of making one chair exceed all others in remuneration. Innumerable instances could be given in which the dead hand which, when living, seemed to be wise has proven in the course of time most unwise.

Another topic which deserves consideration deals with the question of obtaining additional revenue without putting the College in the position of using its building for profit and so falling into the classification in which taxation is levied. In other words, we can legally charge for the use of certain parts of the building at a cost which covers heat, light, attendance and wear and tear. This is now done with medical societies which are not a part of the College, with occasional exceptions, but the Sections have their expenses in practically all points paid for out of the College funds. This is done on the principle, when Sections were started, that a Section was a child of the College and should be nurtured. At present the Sections charge membership fees but the College bears the cost of sending out their notices. The question arises whether the time has not come when the children should contribute a small sum to the support of the mother, at least until such time as the mother, by the sale of property or bequest, is living in comfort. It is with this idea in view that the Library Committee has decided to charge for the use of private study rooms, since heat, light and very considerable attendance is needed by the occupants of these quarters.

Another point which deserves consideration I touched upon in my address of last year, namely, the matter of scientific contributions to the proceedings. At that time I pointed out that very frequently the Committee on Scientific Business had to seek contributions. Perhaps I would speak more truthfully if I said has had to ask Fellows to present papers. This is an unfortunate state of affairs, as it reflects upon the dignity of this body and perhaps results in the presentation of papers unworthy of its position in the medical world. The difficulty may in part be corrected, and it has been so corrected during the past year, by arranging that the lectures under the vari-

ous lectureships be given at the regular scientific meetings. This would not prevent one or two other papers, about which there was any urgency as to time of presentation, from being also on such a program or of allowing papers to accumulate and having them on nonlectureship evenings. Indeed the question might be asked why not have so-called scientific meetings only when Fellows are desirous of giving birth to a worthwhile communication?

Closely allied to this matter is the one which requires that scientific meetings shall be held on the first Wednesday in each month. Perhaps it would be better to let the Committee on Scientific Business call a meeting when it had a program ready and at a date when Fellows could or would attend. For example, the scientific meetings in the late spring often conflict with the meetings of special societies in other cities and this results in a woful lack of attendance. This is also true at other times of the year. Thus at the meeting in October, 1927, when a very able paper was presented by an invited guest who came three thousand miles at our invitation, a mere handful of auditors was almost lost in the hall, and the Fellow who was to open the debate, and who had been chosen because he was considered most likely to add to the interest of the meeting, was absent because of the meeting of the State Medical Society elsewhere. Had the Committee on Scientific Business been able to call the meeting at another approximate date this distressing result could have been avoided. This incident was not unique, and fearing that a similar result might occur at the November meeting the President invited the senior students of two medical schools of this city to attend. The hall was filled, which gave the reader of the paper a good audience; but I may add, *sotto voce*, that the number of Fellows present was quite limited, yet the subject was a very practical one, of interest to every member of the profession. At present a Fellow has a better audience in some instances if he appears before a Section than if he does so before the College itself, and possibly chooses the Section for this reason.

The College, when it permitted the formation of sections, intended that certain rules should be adhered to. One of these is (Rule 6): "No person not a Fellow of the College shall attend the meeting of a Section except by specific invitation to that meeting, by a Fellow, who shall give the name of the visitor to the Clerk of the Section." I believe this rule is never adhered to. Another rule is that the College shall not be subject to any other expenditure than opening,

lighting and heating, but the custom has arisen of having the College as I have stated, pay for the printing and dispatch of the programs. Neither is the President of the College expected to preside at Section meetings if he is present, although the rule states that he shall do so (Rule 3). Apparently it would be well to revise these rules.

We come now to another factor of possible interest, namely, that although there is a rule, as in a Section, that only Fellows and invited guests may be present, there is no such rule that persons, other than Fellows, may not attend the scientific meetings of the College; nevertheless, a general opinion prevails that non-Fellows are not welcome. The breaking of the rule (6) as to the Sections and this feeling in and out of the College as to its scientific meetings militates against the College itself. This unwritten rule as to non-Fellows probably has its origin in the fact that many years ago reporters for the daily press came to the College meetings and reported the proceedings in the daily papers in an objectionable manner, or so it was thought at the time, with the result that a rule was passed that no one except a Fellow might attend a meeting of the College, unless he was first presented to the presiding officer, or, later, unless he presented himself with a card of introduction from a Fellow to that officer before the meeting. Further, this rule was ordered to be printed, if I recall correctly, on all notices of meetings of the College. This kept away a few reporters, who might have slipped in anyhow, and excluded many worthy practitioners who might otherwise attend and be benefited. The question arises, therefore, whether the Sections be made to obey the rules if they invite outsiders or if it would be well for the College to do what the Sections do, namely, state on the notices that members of the profession are invited to attend. Now that the business of the College, as to its private affairs, is taken up chiefly at special business meetings, there is less objection to this than when private business could be taken up at the close of any meeting.

It has been customary when an invited lecturer, under one of the special lectureships, has presented his address for no discussion to take place. I would suggest that such discussion, if permitted on some occasions, would make the Fellows feel that they had the privilege of debate, and often questions might be asked, or opinions expressed, that would add greatly to the interest of the evening.

Three memoirs of deceased Fellows were read during the past year (1927): Of Dr. Henry R. Wharton, by Dr. John H. Jopson;

of Dr. John B. Roberts, by Dr. William E. Robertson; of Dr. Allen J. Smith, by Dr. Edward Martin.

During 1927 I have to record with regret the death of the following Fellows:

Dr. William M. Sweet, died December 24, 1926. Elected June 1, 1898. A memoir read before the Section on Ophthalmology has been deemed sufficient. He made many noteworthy contributions to his specialty, especially as to the localization of foreign bodies in the eye.

Dr. Milton B. Hartzell, died April 7, 1927. Elected October 3, 1888.

Dr. E. E. Montgomery, died April 17, 1927. Elected October 4, 1882.

Dr. E. Tillson Ward, died April 20, 1927. Elected November 2, 1910.

Dr. John G. Clark, died May 4, 1927. Elected April 5, 1899.

Dr. Frank Woodbury, died May 24, 1927. Elected April 7, 1880, and, therefore, a Fellow for forty-seven years.

Dr. Joseph M. Spellissy, died July 27, 1927. Elected November 6, 1895. Active in his specialty of orthopedic surgery.

Dr. Clarence Hoffman, died September 21, 1927. Elected May 7, 1913. An earnest and faithful teacher of anatomy.

Dr. Guido Baccelli was a distinguished foreign Associate Fellow whose work in medicine is well known. He was elected in 1890.

Memoirs, by direction of the Censors, have already been presented at this meeting of Dr. E. E. Montgomery, Dr. John G. Clark and Dr. Milton B. Hartzell.

Since June 30 the old College building at Thirteenth and Locust Streets has been vacant, the Free Library of Philadelphia having moved to its new quarters on the Parkway.

It is of importance to note that a large sum of money will come to the College when the old College building is sold. For some time that particular section of real estate was in little demand, but it would now appear that it is again increasing in value and it is thought that a sale may be consummated in the near future which will be advantageous.

It is the duty of the President at this time to present a brief survey of the work of the various committees of the College. They embody the following facts:

The report of the Secretary, Dr. Girvin, shows that nine stated

meetings were held during the past year and that the average attendance of Fellows was 37. Sixteen papers were read and eleven were illustrated by lantern slides.

On April 6 the College celebrated the centenary of the birth of Lord Lister, an address being made by Sir Esme Howard, the British Ambassador, and another by Dr. A. P. C. Ashhurst.

At the business meeting of January 10 the following amendment to the By-Laws was carried. A new paragraph in Chapter IV, after paragraph 6: The President may at his discretion convene a business meeting of the College at the close of any scientific meeting provided notice shall have been given on the call for the scientific meeting and the necessary quorum is present.

Twenty-three propositions for Fellowship were acted upon; twenty were reported upon favorably, one was postponed and two failed to receive favorable recommendation.

On December 1, 1927, the total Fellowship was 523, of which 502 were Resident Fellows, 15 American Associate Fellows and 6 Foreign Fellows.

The report of the Treasurer has been made to the Council and will be duly audited.

The Committtee on Publication reports that Volume XLVIII for 1926 was distributed in September of 1927.

The Library Committee reports the total number of bound volumes in the Library 154,293. The number of unbound theses and dissertations 29,081, the number of unbound pamphlets 163,064, and manuscripts 431.

The Mütter Museum has been used during 1927 more than ever before for teaching purposes and is constantly improving in equipment and efficiency.

Various necessary repairs have been made by the Hall Committee, which reports continual saving from the installation of the oil heating system.

The Directory for Nurses has registered 1066 nurses. The total number of calls filled was 8256 and a profit balance has been turned over to the College.

Under the direction of the Committee on Entertainment, the Weir Mitchell dinner was given in December.

Under the direction of the Committee on Public Health and Preventive Medicine the Anders Lecture was delivered by Dr. C. E. A.

Winslow, of New Haven, on "The Relation of Indoor Atmospheres to Human Health and Comfort."

The Alvarenga Prize was awarded to Dr. Emil Bogan, of Cincinnati, Ohio.

Two lectures were delivered under the Mary Scott Newbold Lectureships in 1927.

The reports of the transactions of the various Sections have been received by the Council and show that during the year they have been active and successful.

The detailed statements of the various officers and committees of the College have been filed with the Secretary and may be examined by any Fellow who may so desire.

These various statements show that the College during the last twelve months has continued to fulfil the objects of its founders and to be of real service to the Fellows and to the community at large.

It is the wish of the retiring President to once more express to the Fellows his sense of the honor conferred upon him when he was elected to this office, and, further, his appreciation of the courtesy and kindness which has been shown him in the three years while he has held office.

THE CAUSE AND PREVENTION OF THE INCREASINGLY HIGH MORTALITY IN THE ACUTE SURGICAL ABDOMEN*

By JOHN O. BOWER, M.D.

Mortality from appendicitis is increasing. Foreign as well as national statistics substantiate this fact. In England and Wales the increase during the years 1913 to 1923 was 7.3 per cent (total registration over 40,000,000). In 1913 in the United States (total registration over 87,000,000) the death-rate was 12.1 per 100,000; in 1923, 14.8 per 100,000, an increase of 22.3 per cent. In Philadelphia there was an increase of 18 per cent between the years 1915 and 1923 inclusive. At the Samaritan Hospital there has been an increase of 16 per cent over the same period of time.

The increase in the death-rate from appendicitis is 9 per cent greater than that from cancer during the past ten years; and while the total death-rate from cancer is six times that of appendicitis, four times as many people die from appendicitis before the age of fifty years, which is the productive period, than from cancer.

If one were to ask the average physician today if the operation for appendicitis is a serious one, he would tell you that the mortality is practically *nil*. While this is true of subacute and chronic cases, it is surprising to know that in the average hospital in the United States, 1 out of every 12 patients who enter the hospital with a diagnosis of acute appendicitis dies. The morality varies from year to year. Some years it may be as low as 3 per cent; in others it will exceed 8 per cent.

Opinions differ as to the cause of this increase in mortality. There are surgeons who believe that surgical technic and management of the patient after admission to the hospital are at fault. In England, Cecil Rowntree[1] states that "the Registrar-General's figures, which showed an increase in mortality, were accounted for by the number of inexperienced surgeons now operating and by their persistence in removing the appendix in cases where it should

* Read January 5, 1927.

[1] Proc. Roy. Soc. Med., 1925, vol. xviii, No. 2.

be more safely left." In the United States, Willis[1] thinks that it is due to lack of standardization of methods of treatment, and suggests that a commission composed of leading surgical teachers of the country be appointed to direct a thorough investigation. While it is realized by all surgeons that early operation is the ideal for which to strive, few have suggested a remedy. In Canada, however, E. M. Eberts and L. H. McKinn[2] write that "the remedy lies through the joint action of the members of such a body as the Canadian Medical Association."

I hope to show in this review of 1010 cases of the acute surgical abdomen that the cause of the high mortality-rate, for the most part, is due to loss of time between the onset of symptoms and operation, and that the prevention is the education of the public through the family physician. I have included six acute lesions of the abdomen which are decidedly surgical. In the order of their importance they are appendicitis, intestinal obstruction, acute perforation of the duodenum or stomach, acute pancreatitis, ectopic pregnancy and acute perforation of the gall-bladder.

Ectopic Pregnancy. In no acute surgical lesion involving the contents of the abdomen is the mortality as low as in ectopic pregnancy. Nature's provision for the perpetuation of the species is here superbly demonstrated. In no other part of one's anatomy can one lose so much blood, be operated upon and live. Post-mortem examinations on patients operated on for gall-bladder disease revealed that blood not exceeding 500 cc. in amount escaped from the cystic artery and was apparently the cause of death; yet the same amount of blood, of ovarian or tubal origin, has been observed in the peritoneal cavity of a young woman with few symptoms and signs to denote its presence. The time between onset of symptoms and operation has a bearing on the mortality in ectopic pregnancy. There have been but 2 deaths in this series of 63 cases, no mortality in those entering the hospital within twenty-four hours and a mortality of 3 per cent in those entering after that period of time.

Perforation of the Gall-bladder and Acute Pancreatitis. These will be considered together. Etiologically they are directly dependent upon pathologic changes in the gall-bladder, which in most instances have been present for a long period of time. Acute

[1] Surg., Gynec. and Obst., 1926, xiii, 321.
[2] Canada Med. Assn. Jour., 1921, xi, 443.

perforation of the gall-bladder with associated generalized peritonitis is most unusual. In a review of 914 charts of patients operated upon at the Samaritan Hospital for gall-bladder disease, I found that 10 had perforated. Of this number 2 had perforated into the general peritoneal cavity; 1 of these died. There was no mortality in the subacute cases. Less than one-quarter of 1 per cent of patients required emergency surgical treatment. However, 2 per cent of all gall-bladders operated upon had an associated acute pancreatitis. Combining the cases of gall-bladder perforation with acute pancreatitis, both requiring prompt surgical intervention, we have a total of 27, less than 3 per cent of all gall-bladders operated upon. While this percentage is small and relatively insignificant when compared with the first three acute lesions mentioned, it must be reckoned with. We can, however, deal with them in comparative safety since in this series the patients enter the hospital within an average of thirty-four hours for perforated gall-bladder and thirty-six hours for acute pancreatitis. Medical measures (large doses of morphine, external applications, etc.) having had no effect, the patient's insistence on relief from pain resulted in early hospitalization and surgical treatment. While the foregoing statement is a reflection on our treatment of these cases, its truth is borne out by the facts I will present to you regarding the management of acute appendicitis, intestinal obstruction, and perforation of the stomach and duodenum.

Acute Perforation of the Stomach and Duodenum. There is no pain comparable to the pain of perforation of the duodenum with the possible exception of acute pancreatitis. The patient frequently rolls on the floor or writhes in bed, in agony. In most of these cases there is a state of severe shock because of the proximity of the lesion to the solar plexus. Moynihan reports a case where the shock accompanying the perforation was so severe that death was instantaneous. In these cases, morphia, the panacea, the smoke screen behind which hovers the Destroyer Death, unless given in large doses, has no effect and the patient reaches the hospital early. In this series, 1 patient was operated upon two and a half hours after perforation, the average for the 52 being 32.3 hours; for the recoveries 23.5 hours, and the deaths 56.1 hours. The early symptoms of acute appendicitis and intestinal obstruction are usually more subtle than those of perforation of the stomach, duodenum or gall-bladder or of an acute pancreatitis.

Acute Appendicitis. In this series of 750 cases of acute appendicitis, 338 or 45 per cent perforated before entering the hospital. One hundred and sixty-three, or 48 per cent of all perforations and 21.7 per cent of all patients operated upon, had perforated into the general peritoneal cavity. While in the greater percentage of instances a diagnosis of appendicitis was made by the attending physician, less than 1 per cent entered with a diagnosis of perforation. The gravity of this observation can be appreciated when we realize that 43, or 71.6 per cent of the 60 deaths had perforated.

You will observe that in the recoveries the average time between onset of symptoms and operation is 69.1 hours. The average time between onset of symptoms and operation in the perforated cases was 107.3 hours. The time element is the all-important factor. Comparing our minimum mortality which is 1.53 per cent in 1923 with our maximum 9.6 per cent in 1922, we find there is an increase of 72 per cent in the time which elapsed between onset of symptoms and operation in 1922; in 1922 there were 100 per cent more perforations and while this has a bearing on the increase in mortality, it is not so constant a factor as the time element; for example, in 1925 with a mortality of 8.82 per cent, a 576 per cent increase over 1923, we have the same percentage of perforations as in 1923 but an increase of 47.4 per cent in the time between the onset of symptoms and operation.

It is the opinion of many that pathological changes occur in appendices with little or no pain; certainly this is not true of a perforation into the general peritoneal cavity, and while the pain of a perforated appendix is not as severe as that of a perforated duodenal ulcer or an acute pancreatitis, it is so severe that these patients invariably seek medical aid immediately. One out of ever 5 patients perforates into the general peritoneal cavity and is beyond the aid of anything other than operation when first seen by the attending physician. This group is responsible for 73 per cent of our mortality. Fifty-two per cent of perforations are taken care of by the defensive mechanism within the peritoneal cavity which accounts partly for the delay in the patient's entering the hospital and in part for the management by some physicians. Fortunately the percentage of men in our ranks who persist in treating abdominal pain as though it in itself were a distinct entity rather than a warning of impending danger, is gradually diminishing.

Intestinal Obstruction. We have combined hernial and internal obstruction of the intestine. As would be expected, the average time between onset of symptoms and operation is much less for hernial than for internal obstruction; thirty-seven hours for hernial and 76.6 hours for internal obstruction, the latter being 177 per cent greater than in hernias. Combining both types of intestinal obstruction gives us a total of 130 cases. Of these the deaths show 120 per cent increase over the recoveries in hours from onset of symptoms to operation.

We can divide the definitely acute surgical abdominal lesions into two groups. In the first group, ectopic pregnancy, perforated gall-bladder, perforated stomach and duodenum and acute pancreatitis. Nature will, in the majority of instances, take care of the bleeding from a ruptured ectopic pregnancy. An individual suffering with a perforated gall-bladder, duodenum or stomach, or acute pancreatitis will demand relief unless his symptoms are masked with morphia. The second group, acute appendicitis and intestinal obstruction, warrants our serious consideration. (1) Because of the insidiousness of the onset; (2) because of the patient's delay in calling a physician; (3) because of the frequency with which laxatives have been administered; (4) because of the delay due to the physician's temporizing with the patient and the patient's family.

I am in thorough accord with Dr. Cope who says, "The general rule can be laid down that the majority of severe abdominal pains which ensue in patients who have been previously well and which last as long as six hours, are caused by conditions needing surgical intervention."

It is within the domain of the family physician to teach the public, but before he can teach he must take a definite stand; he should not institute medical measures and he should refuse to assume the responsibility for a patient's life in the presence of persistent acute abdominal pain. If he is broad-minded enough to suggest to his patient that a surgeon share this responsibility, the mortality in the acute abdominal lesions will be diminished.

Ectopic Pregnancy—65 Cases

	Cases.	Deaths.	Mortality, per cent.
Under twenty-four hours	18	0	0.00
Over twenty-four hours	47	2	4.1

Acute Pancreatitis—13 Cases

Average time—onset symptoms and operations . . Recovery 27.7 hours; Deaths 63.0 hours

	Cases.	Deaths.	Mortality, per cent.
Under twenty-four hours	10	0	0
Over twenty-four hours	3	2	66.67

Acute Perforation of Gall-bladder

Total number of pathologic gall-bladders operated upon	914
Total number of perforations—local peritonitis	8
Total number of perforations—general peritonitis	2
Total number of deaths—perforation	1

Perforated Duodenal Ulcer—52 Cases

Average time—onset symptoms and operation . . Recovery 23.5 hours; Deaths 56.1 hours

	Cases.	Deaths.	Mortality, per cent.
Under twelve hours	23	1	4.35
Under eighteen hours	29	2	6.9
Under twenty-four hours	35	4	11.4
Under forty-eight hours	45	9	20.0
Under seventy-two hours	47	11	23.4
Over seventy-two hours	5	3	60.0

Intestinal Obstruction—129 Cases

Average time—onset symptoms and operations . . Recovery 36.1 hours; Deaths 83.7 hours

	Cases.	Deaths.	Mortality, per cent.
Under twelve hours	23	3	13.0
Under twenty-four hours	56	5	8.9
Under thirty-six hours	66	9	13.6
Under forty-eight hours	84	22	26.1
Under seventy-two hours	102	31	30.4
Over seventy-two hours	27	17	63.0

Acute Appendicitis—751 Cases

Average time—onset symptoms and operations . . Recovery 69.1 hours; Deaths 151.0 hours

	Cases	Deaths.	Mortality, per cent.
Under twelve hours	49	2	4.0
Under twenty-four hours	225	3	1.3
Under thirty-six hours	308	5	1.6
Under forty-eight hours	438	13	3.0
Under seventy-two hours	560	30	5.32
Over seventy-two hours	191	30	15.7

Year.	Mortality.	Time of sickness and oper. gen., per hours.
1912–1914	4.00	71.0
1916–1920	6.35	82.0
1921	0.00	36.0
1922	9.60	85.2
1923	1.53	49.5
1924	1.70	54.5
1925	8.82	73.0
1926	5.3	60.5

This table shows the relationship which exists between mortality and time between onset of symptoms and operation in patients entering the hospital with a diffuse peritonitis; this group being responsible for 73 per cent of our mortality.

The prevention of this increasingly high mortality lies in the reduction of the time between onset of symptoms and operation. This can be successfully brought about by obtaining the coöperation of the staff and physicians who refer patients to the hospital. We have done this at the Samaritan Hospital by sending out a letter emphasizing the following points:

1. The importance of diminishing the time between onset of symptoms and operation.

2. Public education through the family physician relative to the use of laxatives in the presence of abdominal pain.

3. Time element affects the prognosis but has no bearing on the character of the pathology. Gangrene may occur as early as five hours and perforation as early as twelve hours.

A follow-up letter will be sent out every three months reporting progress.

	Before letter.		*After letter.*	
	Recovery.	Deaths.	Recovery.	Deaths.
Intestinal obstruction . .	38.0 hours	83.7 hours	35.0 hours	0.0 hours
Ectopic pregnancy . . .	24.0 hours	47.0 hours	24.0 hours	0.0 hours
Perforated duodenal ulcer .	23.5 hours	56.1 hours	10.0 hours	0.0 hours
Appendix	65.9 hours	151.0 hours	42.6 hours	40.6 hours

SYPHILITIC SPLENOMEGALY ASSOCIATED WITH AN OSTEOMYELITIS OF THE CLAVICLE*

By THOMAS KLEIN, M.D.

In reviewing the literature one is struck by the comparative infrequency of the above associated lesion. For the past eighteen months we have had under our care the following case:

J. P., aged thirty-eight years. Vertical headache. The patient was well up until January, 1925, when he began to complain of headache on the top of his head. He has had the pain in his head almost constantly ever since.

He has been advised to get glasses, which he did, but has obtained no relief. At times he has little pain in lumbar spine. His appetite is good with no postcibal disturbance. His bowels are regular. At times he has a little burning on urination and some increased frequency with nocturia at times. He has no cough or expectoration; no dyspnea; no precordial pain; no cardiac palpitation and no dizziness. The head pain does not interfere with sleep but he tires out very readily. He has lost little weight. He does not remember having had childhood diseases. He had gonorrhea in 1908; chancre in 1909 and influenza in 1918. He gets sorethroats and tonsillitis. He has had no operations.

The patient is a tailor. His eating habits are good; drinking but little coffee and smoking lightly.

His wife and six children are living and well. There is no history of diabetes in his family. His brother died of tuberculosis.

Physical Examination. Blood-pressure: 120 systolic, 60 diastolic; pulse, 100; weight, 140 pounds. The patient is a well-developed and fairly well-nourished adult, male, Italian. Color is good. Station and gait normal.

Ears and Nose. Grossly negative.

Eyes. Pupils react well to light and accommodation. Palpebral conjunctiva, good color.

Mouth. Teeth rather poor. Tongue slightly coated. Tonsils are diseased, cryptic. Marked *fetor ex ora.*

Neck. Slight cervical and submaxillary lymphadenopathy.

Chest. Normal. Tinea versicolor.

Heart. Left border just outside nipple line. There is a soft systolic murmur at apex. A_2 greater than P_2.

Abdomen. There is some definite fulness in left upper abdomen. On palpation a definite resisting mass is felt. The mass extends through

* Read January 5, 1927.

entire left hypochondrium, 3 cm. below the umbilicus, and to the midline of the abdomen. The mass is smooth and firm. Not tender or sore. The liver edge is palpable on inspiration. The edge is sharp with no areas of tenderness. There is no fluid in abdomen.

Right Clavicle. The right clavicle is markedly enlarged and not tender. There is no evidence of any other periostitis.

Extremities. The reflexes are prompt and active; no edema; no inguinal, axillary or epitrochlean lymphadenopathy.

Laboratory Reports. *Urine.* Specific gravity, 1.015; reaction, acid; albumin, negative; sugar, negative; two to three white blood cells, high power field. Mucus +.

Blood Count. Hemoglobin, 70 per cent; red blood cells, 3,660,000; white blood cells, 5300.

Differential Count. Polymorphonuclears, 89, 75; lymphocytes, 6, 20; large mononuclears, 1, 2; transitionals, 3, 3; eosinophils, 1

Blood Chemistry. Blood urea nitrogen, 16 mg.; blood-sugar, 68 mg.; blood uric acid, 2.3 mg.; blood creatinine, 1.1 mg.

Wassermann. +4.

Gastric Test.	Free HCL.	Total acidity.
	10	35
	20	60
	35	60
	30	55
	40	60
	30	60

Occult blood, negative.

Feces. Occult blood, positive.

Diagnosis. 1. Tertiary syphilis. (*a*) Interstitial splenitis; (*b*) syphilitic osteomyelitis of clavicle.

A few days after our first examination, while waiting for the report of the blood Wassermann reaction, the patient again presented himself, with a spontaneous fracture of the clavicle. This occurred while getting on a street car. It was absolutely painless. The *x*-ray report by Dr. H. K. Pancoast's laboratory is as follows:

The *x*-ray examination of the right shoulder shows a pathological fracture of the outer and middle third of the clavicle. There is no evidence of bony union. The appearance of the clavicle, as a whole, is that of a luetic osteomyelitis.

The arm was strapped to his body and healed readily under antiluetic treatment.

In a study of the literature and case reports of syphilitic splenomegaly one would be led to believe that an interstitial splenitis of this type is practically unresponsive to our present methods of treatment. In most instances there was little or no reduction in

size of the spleen, even after so-called intensive antiluetic treatment, using arsphenamine, iodides, and mercury. Mercury seemed to give the best results. In not one case have we been able to find a report of a change in the blood Wassermann reaction after such treatment. Splenectomy was resorted to finally in many instances, with variable results.

In the vast majority of cases where splenectomy was done the antiluetic treatment had not been sufficient to allow for retrogressive changes in the spleen in lieu of our present knowledge of the arsphenamines. Most cases were under observation rarely more than six months before resorts to surgery. This patient has been under constant treatment for eighteen months. During this time he has received 40 intravenous injections of neoarsphenamine of 9 decigrams each and 7 intramuscular injections of potassium bismuth tartrate at weekly intervals. At the end of this time he developed a bismuth line and it was thought advisable to discontinue. Since then he has been given 10 intramuscular injections of mercury salicylate. He also has been taking large doses of iodides from the start. The spleen was reduced over one-half in size. It now extends 3 cm. below the costal border. It is much less firm and not at all painful. The patient has lost all his subjective symptoms and is potentially well. The headaches entirely disappeared and the clavicle healed. However, the blood Wassermann still continues to be strongly positive. This finding is in accord with practically all reports. We doubt very much whether there will be further reduction of the spleen in size because of the interstitial change in that organ.

I. Syphilis of the Spleen. Syphilis of the spleen, according to Wile,[1] is common in congenital, but rather uncommon in acquired types. It is more frequent in the early stages, occurring coincident with a general lymphadenitis. It may be overlooked because of the usual insensibility of the splenic tumor. Syphilis of the spleen may occur: (1) Early, as an acute splenic tumor, with or without pain; (2) late, as an interstitial disease. In such cases where there is a syphilitic disease of the liver, accompanying the enlarged luetic spleen, the pathological condition may simulate Banti's disease.

Early Syphilis of the Spleen. Biermer in 1862 noted that splenic enlargement is associated with the earlier manifestations of syphilitic infection. In 1874 A. Weil published observations on splenic hypertrophy in acute syphilis, and found that splenic tumor was present in 36 per cent of the cases studied. Of the 36 cases

studied, 19 were found to be soft, with rounded edge, as in typhoid fever. Others were firm and readily palpable, similar to those cases of interstitial splenitis of chronic malaria. Fourteen cases showed a leukocytosis of over 10,000. The splenomegaly in early cases is usually symptomless, and the anemia and digestive disturbances, which may be present, are probably due to the constitutional infection. Under intense antiluetic treatment the spleen rapidly decreases in size, and, if untreated, goes into the form of an interstitial splenitis.

Late Syphilis of the Spleen. 1. *Interstitial Splenitis.* Interstitial splenitis is seldom found *per se*, but is usually associated with syphilis of the liver, kidneys, and other viscera. Except for its size there are usually no clinical symptoms. On palpation the spleen is found to be firm, resistant, and occasionally slightly tender. Gastrointestinal symptoms are usually associated with syphilis of the liver. According to Wile the prognosis of the disappearance of the splenic tumor is not good.

2. *Gummatous Splenitis.* This condition is common in syphilis of the newborn, but rare in acquired syphilis. The gummas may be from the size of a pea to that of a walnut, or may exist as miliary gummas. This condition is only discovered at necropsy.

3. *Amyloid Disease of the Spleen.* This form of splenitis is recognized clinically as such in the presence of amyloid changes in the intestines or in other viscera. Usually it is a pathological entity rather than a clinical one.

Turno[2] has reported 3 cases of enlargement of the spleen in syphilitic women, with fever in 2 of them. One of these patients exhibited a typical Banti's disease in the secondary stage. Under treatment of the syphilis the clinical and pathological manifestations subsided completely. He differentiates this condition from true Banti's disease by the presence of an intermittent type of fever, which is often preceded by a chill, and by a positive blood Wassermann reaction. According to him the spleen is not so large as in Banti's disease, and is more painful. He states that syphilitic women are particularly prone to show splenic disease. Specific treatment is usually effectual in all phases of the disease, and if not, splenectomy gives brilliant and durable results.

Timbal[3] treated a man, aged sixty-five years, with splenomegaly of luetic origin, by using mercury. He states that the spleen was reduced to normal size under this form of treatment. He cites

9 other cases that had been published in France, all showing the prompt efficacy of mercurial treatment.

I. W. Held[4] states that the spleen may become very large in acquired syphilis. It may be accompanied by a secondary anemia, a leukopenia and a mononucleosis. The mononucleosis was not present in our case. Under such conditions it is readily mistaken for Banti's disease until a positive blood Wassermann test proves otherwise. In fact, until the era of the Wassermann test, Banti classed these cases as such belonging to the group described by him. A luetic splenomegaly, with anemia and ascites, Held states is a more progressive type of disease than is the true Banti's disease.

Faucett[5] reported a case of a man, aged twenty-five years, with splenomegaly, enlarged liver, jaundice, and a positive blood Wassermann reaction. The blood shows a leukopenia with a normal differential count. On several occasions the patient vomited blood and passed blood by bowel. He was placed on mercury and ascending doses of iodides over a period of six months with an occasional short interval of freedom from treatment. The patient also received 6 intravenous injections of novarsenobenzol. After this treatment there was very little diminution in the size of the spleen, and the Wassermann remained strongly positive. He feels that the ultimate prognosis in these cases is not good.

Emile Weil[6] has reported the blood Wassermann reaction in various forms of splenomegaly. Several cases of leukemia and chronic splenomegaly were reported with positive tests. The reaction of the blood was not changed in any after intensive antiluetic treatment. There was little or no diminution in the size of the spleen in any of the cases. The general symptoms were often aggravated. He believes that these cases are not actually syphilitic, for no trace of hereditary or acquired syphilis could be determined, and that they may be due to some unknown parasite or bacterial organism.

In any type of splenomegaly it is very essential to determine the blood Wassermann reaction. If found to be negative, such conditions as malaria, splenic anemia, Banti's disease, leukemia, cirrhosis of the liver with secondary splenomegaly, malignant endocarditis, or Hodgkin's disease must be considered. Erythremial (Vaquez's disease), tumors, abscess, tuberculosis, Gaucher type of splenomegaly, and kala-azar must be borne in mind.

II. Syphilis of the Bone. Goodman[7] has recently made a careful review of syphilis of the bone. In congenital syphilis the pathological bone manifestations are usually around the epiphyses. In acquired syphilis there may be local or diffuse changes more or less limited to the periosteum, or to the bone itself. Periostitis of the bones, particularly the frontal, may occur as early as thirty-four days after the appearance of the chancre, according to Mauriac. Suchanek stated that in Prague bone syphilis was seen in 7 per cent of the cases during the primary stage. Clinically the patients complain of dull pain over the bones, particularly the superficial bones. Pain is usually more pronounced at night. Wile and Senear[8] state that the spleen and bones are often pathologically involved in early syphilis. The general health is severely affected, as is shown by the severe anemia, debility, and loss of weight. In about 30 per cent of the cases in which the bones and joints were involved they found definite splenic enlargement. Most of them had pain. The pain was mostly nocturnal in character, and distinctly medullary in type. In their experience such pain is not so readily amenable to treatment, as is that due to periostitis. Objectively periostitis is the most frequent clinical finding. The tibia, sternum, skull, and ribs are most frequently involved. The clavicle was the only site in which periosteal node was painless. Pain in the joints, like pain in the bones, is relieved by exercise. Whenever arthralgia was present, the symptom was soon relieved after efficient treatment was begun. They believe that there may be so-called "spirochetal rests" in the bone-marrow, and if the bones are traumatized they may serve as distributing foci of the later sequelæ and may recharge the circulation with spirochetes.

Goodman[7] states that syphilis of the bones is comparatively uncommon in the secondary stage. Although any bone may show the lesion, certain ones are more frequently involved. Popescu gives the following table:

Frontal bone	28 per cent
Ribs	14 "
Tibia	16 "
Parietal	9 "
All others	8 "

During the tertiary period of lues, changes occur more frequently. These lesions may manifest themselves from four years to forty years after the appearance of the chancre. Jullin gives this table of the relative frequency of bones affected in tertiary lues:

Nose	30.0 per cent
Tibia	22.0 "
Palate	21.0 "
Sternum	21.0 "
Clavicle and maxillæ	3.0 "
Frontal, parietal, vertebræ of each	1.5 "
Scapula, ulna, patella of each	1.5 "

The nasal bones necrose and leave the saddle nose deformity. The palate often perforates. A perforated nasal septum is always to be considered luetic until proved otherwise. In congenital or acquired luetic dactylitis the phalanges are the site of gummatous osteoperiostitis, with enlargement and rarefaction of the bone. The prognosis of bone syphilis is none other than that of syphilis of the individual. Scars may remain, but lues of the bone is definitely amenable to treatment.

Gomez[9] reports 3 cases in which an acute osteomyelitis developed in a bone already the seat of a syphilitic process. He states that he had not found this combination mentioned in the literature.

In a tertiary bone syphilis, says W. P. Sights,[10] we may have superficial or deep gummatous types of lesions. Superficial gummas appear between the cortex and periosteum of the bone, with ulceration and subsequent roughening. Deep gummas may appear in any part of the bone. At times they appear in the spine simulating a Pott's disease. Gummata of the sternum, ribs, and clavicle may be found.

Comment. The combination of visceral and bone syphilis is comparatively rare in the tertiary stage. In syphilitic splenomegaly the spleen can be reduced greatly in size by intensive and prolonged antiluetic treatment. Splenectomy should not be resorted to as long as the patient improves under this régime. The blood Wassermann remains unchanged even with the patient potentially well.

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DISCUSSION

Dr. Edward B. Krumbhaar: Syphilitic splenomegaly has always had for me two points of interest, both of which Dr. Klein mentioned: first, its frequent resistance to antisyphilitic treatment, and, second, its possible confusion with Banti's disease. In many cases there is very apt to be a leukopenia and distinct anemia associated with the enlarged spleen, in fact, this seems to be a common accompaniment of many chronic enlargements of the spleen. Therefore, in the chronic tertiary stage it becomes so difficult to make a diagnosis that many pathologists, notably Warthin, consider there is no such disease as Banti's disease, *per se*, but that it can always be relegated to some other category, and particularly syphilis, which may involve the splenic artery or vein with secondary changes in the organ itself, again giving signs and symptoms practically indistinguishable from Banti's. I feel, however, that such cases should be considered "pseudo-Banti's Disease" and that it is possible to distinguish by careful clinical and pathological study a nonsyphilitic disease primary in the spleen that answers to Banti's description.

The other point is even more interesting, namely, the resistance of syphilitic splenomegaly to antiluetic treatment. Dr. Klein's case is important because he continued treatment so long; but in syphilis, as in malaria, the spleen may assume a chronic condition, like the parasyphilitic central nervous system, wherein the treponema cannot be reached by the ordinary methods of antiluetic treatment, and I think the persistence of the positive Wassermann reaction has indicated that this may be the case here. If that is true, it would seem that splenectomy might still be considered after the patient has no longer continued to improve under the antiluetic treatment. I have been interested in trying to follow the results of treatment in splenectomy for many years. In the syphilitic cases in which it has been tried not more than a dozen—there has not been more than one death and all the others were markedly improved. It may be, as Dr. Klein says, that in some of these the antiluetic treatment was not carried out long enough; but I still think that this condition should be considered a proper field for splenectomy after the other measures have failed.

Dr. W. Egbert Robertson: Not long ago you may recall that in *The Journal of the American Medical Association* there appeared an editorial calling attention to the frequency with which enlarged spleen is encountered in anemia. For years it has been overlooked, for there is nothing in the literature about enlarged spleens. I believe that in the secondary stage

of syphilis the spleen is always enlarged. I think where it is carefully palpated and percussed it can be always demonstrated that it is much like anemia in the sense it has not been looked for. The real splenomegaly is the exception. It may be looked for in the congenital. About fifteen years ago I had a patient in my ward at the Episcopal Hospital, a girl, twelve years of age, who had an enormous spleen and also enlargement of the liver. She had a mononuclear increase with a moderate decrease in the total count; she had a positive Wassermann. The mother's Wassermann was negative, the father's was 3 +. Treatment was instituted and the girl made a very excellent recovery and both liver and spleen returned to normal size, but I made a Wassermann of her blood within the last six months and it is still double plus, which is rather interesting in connection with Dr. Klein's subject.

Another interesting thing in connection with the case is the history of the father, who was admitted to the Episcopal Hospital with an abdominal tumor, for which operation was advised; but, curiously enough, on that day sugar was found in his urine, so operation was deferred. He was transferred to the medical side. His Wassermann at that time was negative, though seven years before it had been strongly positive; now it was negative, but the tumor was not operated on. He was turned over to the medical side, and it was thought there to be a gumma, and appropriate treatment was instituted and the tumor entirely disappeared. The interesting thing is that four years later he developed an attack of apparent obstruction of the bowel, was operated on at the Samaritan Hospital and we found a constricted mass of scar tissue containing typical luetic tissue, which was the end-result of the tumor which he had had four years before, which was a gummata.

Another interesting thing in connection with the case: The child recovered and is now living, twenty-seven years of age, and in apparent good health. She was one of three children; the first died at birth, the second lived to be two years of age, this child was twelve years of age when she was admitted to the ward.

INFECTION WITH TUBERCULOSIS IN CHILDREN AND IN ADULTS, AND THE RELATION OF ONE TO THE OTHER

The Mary Scott Newbold Lecture
Lecture XVII*

By EUGENE L. OPIE, M.D.

In the early part of the Eighteenth Century drastic laws to control the spread of phthisis were promulgated by royal decree in Spain. Under penalty of fine, imprisonment and exile, physicians were required to secretly report to a magistrate anyone who suffered or was suspected of suffering with heetic fevers, consumptions or other contagious disease. After death of the patient it was direeted that clothing, bedding and furniture be promptly destroyed by fire; that the plaster of the chamber be picked off and replaeed, that the floor be retiled. Similar laws were enacted with no little opposition in the kingdom of Naples and in Portugal (Fliek).

Casual reference to some of the great names of medicine will show wide difference of opinion among those who have given thought to the eausation of phthisis. Espeeially noteworthy is the statement of De la Boe Sylvius, who first deseribed the miliary tuberele. He says that irritating emanations are continually carried from eonsumptives to those in their environment, so that they beeome infected with the poison, especially in tender and younger years.

Morton, Valsalva, Morgagni and many others have believed that consumption is contagious. Cullen, Portal, Rush, may be cited among those who have found no evidence that the disease is transmissible from the sick to the well. In his book on phthisis, published in 1825, Louis eonsiders in detail the etiology of the disease, but does not mention the possibility of transmission from the affected patient to healthy individuals.

* Read February 2, 1927.

Some of the most illustrious of those who have contributed to knowledge of tuberculosis have regarded the patient with consumption as a source of danger to those about him, but express the opinion that the disease cannot be conveyed by soiled clothing and utensils, but is transmitted only when contact is intimate and prolonged. In this group belong Galen, Laennec and Villemin.

With the discovery of the tubercle bacillus, by Koch, and its demonstration in the sputum of patients with phthisis, the assumption that the disease is transmitted by direct transfer of the microorganism from the diseased to the healthy individual gained wide acceptance and has been the foundation upon which preventive measures and educational propaganda have rested. It is the creed of all associations for the prevention of tuberculosis. An immense amount of inconclusive investigation has been directed to determine in what degree dust of dried sputum, droplets expelled from the respiratory passages and clothing or utensils contaminated with the tubercle bacillus are a source of danger to those who come into contact with them. Measures for the prevention of tuberculosis belong chiefly to two categories: Those directed to stop promiscuous scattering of infected sputum and those directed to segregate the individual who expels the microörganism.

First Infection and Reinfection

No phase of tuberculosis can be adequately discussed unless a distinction is made between the tuberculosis of infancy and early childhood, on the one hand, and tuberculosis of adolescence and adult life, on the other. This distinction is applicable to the etiology, pathology, clinical course and prevention of the disease. Tuberculosis of infants and tuberculosis of adults, though produced by the same agent, differ so widely that we might profitably describe them as though they were different diseases.

There are two outstanding truths concerning the contagion of tuberculosis now widely accepted: Infants are susceptible to infection and their infection often bears an obvious relation to contact with open tuberculosis. The source of contact is usually a parent, but Holt cites the infection of ten infants within fourteen months by a tuberculous midwife who established respiration in the newly born by applying her mouth to that of the infant.

Adults are obviously insusceptible to infection and it is rarely possible to trace with certainty the source of phthisis presumably acquired during adult life. Often quoted are cases such as that of H. Weber: A sailor with consumption had had four wives, and each in succession, healthy at the time of marriage, died with tuberculosis. The disease being widespread, these instances it is claimed should be regarded as coincidence.

The opinion that much adult tuberculosis has had its origin in infancy is supported by the authority of von Behring: Phthisis, he has said, is the end of a song sung over the cradle of an infant. He has supported his opinion by analogies in large part derived from experiments on animals. He has cited experiments, performed by himself and his coworkers, which show that proteins, diphtheria and tetanus toxins, anthrax bacilli and finally tubercle bacilli pass through the intestinal mucosa of the newly born, whereas they have no effect when fed to older animals. Tuberculosis of animals produced by inhalation or by feeding is not followed by cavity formation, and he has succeeded in producing a disease resembling phthisis of human beings only when he has fed microörganisms so attenuated that they have caused a chronic disease persisting during a long period. Demonstration that pulmonary tuberculosis is almost invariably caused by the human bacillus disproves the suggestion that it is derived from tubercle bacilli ingested with milk, but leaves open the possibility suggested by von Behring that tubercle bacilli swallowed with the mother's milk implant the seeds of a disease which matures in adult life. Nevertheless, I shall not further discuss the intestinal origin of pulmonary tuberculosis, for it has at present little support.

Infection of Adults

Evidence concerning the difficulty of infection during adult life has been obtained in great part from two sources. Statistics from sanatoria for the treatment of tuberculosis seem to show that doctors and nurses in contact with the disease do not acquire it more frequently than others not so exposed. An immense collection of statistics are cited to show that tuberculosis is not more frequent among the husbands or wives of consumptives than in the general

population. Pope and Pearson, studying 40,000 couples, seem to have found scant, if any, evidence of marital infection; whereas Arnould, examining the history of 53,000 couples, found some evidence of transmission from one consort to another. Such an eminent authority as Newsholme doubts the accuracy of these statistics and questions if they will determine the frequency of infection during adult life.

The belief that the tuberculosis of adults is acquired in childhood is widespread. One well-known American authority on tuberculosis (Pottenger) says: "Tuberculosis is believed in nearly all instances to be an extension of a prior infection which took place in childhood but failed to heal." The author of a well-known treatise on pulmonary tuberculosis (Fishberg) says: "Infection occurs in nearly all cases during childhood, the bacilli remaining latent within the body until some exciting cause reactivates them, or the natural resistance is reduced and tuberculous disease results by endogenous infection."

Before the last meeting of the International Union against Tuberculosis, Professor Ronzoni, invited to lead the discussion of the communicability of tuberculosis in adults, stated that phthisis may be a late manifestation of a disease contracted in infancy, and although the possibility of infection of adults must be admitted, prophylactic protection and antituberculous vaccination of the infant are the most efficacious means for protecting adults against tuberculous infection.

Shortly after Koch's discovery of the tubercle bacillus, knowledge concerning immunity against tuberculosis began to accumulate. The first step was the discovery of tuberculin and of related phenomena by Koch himself. A little later, knowledge of the relation of human tuberculosis to that of lower animals developed out of Theobald Smith's discovery of the bovine type of tubercle bacillus. Coincident with these discoveries there has been a gradual accumulation of information which has broadened knowledge of the disease. Three important advances may be cited:

1. Realization of the almost universal distribution of tuberculous infection among those who are crowded together in large cities.

2. Recognition of the absence of tuberculosis among aboriginal races who have not come into intimate contact with European civilization and of their susceptibility to acute forms of the disease.

3. Clear understanding of the differences between the tuberculosis of infancy and early childhood, on the one hand, and phthisis of adult life, on the other.

Pulmonary Tuberculosis of Children

The outstanding characters of pulmonary tuberculosis in children may be described as follows: Pulmonary lesions occupy one part of the lung as frequently as another and are accompanied by tuberculosis of those lymph nodes which are nearest in the direction of lymph flow (Figs. 1 and 2). During the first year of life these lesions tend to progress, and death with generalized tuberculosis or with tuberculous meningitis is the usual termination. Occasional exceptions to this statement are met. During the second year of life the disease tends to persist longer, but the tendency to heal is scant. At a later period progressive fatal tuberculosis is less common, and in those who have died with diseases other than tuberculosis are found with increasing frequency as age increases in lungs and regional lymph nodes focal nodules which exhibit fresh caseation, encapsulation with fibrous tissue and every grade of calcification. Some of these nodules, which may be regarded as scars of obsolescent tuberculous infection, contain soft, puttylike material which casts a shadow upon an *x*-ray plate, and others are as hard as stone. They persist throughout life and in the adult prove, as Naegeli first showed, that almost every adult has been infected with tuberculosis during childhood.

In most instances these lesions have remained latent, being unaccompanied by recognized symptoms or discoverable physical signs, but not infrequently healed tuberculous lesions in lungs and tracheobronchial lymph nodes of adults are more extensive (Fig. 3) than some of those lesions which in children have caused acute miliary tuberculosis or tuberculous meningitis. It is evident to the pathologist who gives attention to the tuberculous lesions of those who die from diseases other than tuberculosis, that there is every transition between latent and fatal tuberculous infection.

Pulmonary tuberculosis of children, which invariably implicates the adjacent lymphatic nodes, has the characters of tuberculosis produced experimentally in animals, for example, in the guinea-pig, and pursues a similar course. It has long been recognized that the phthisis of adults has little resemblance to tuberculosis produced

FIG. 1. Roentgenogram of the lungs of a man, aged seventy-four years, who died with general arteriosclerosis and chronic nephritis. There are firmly calcified nodules in the right lung and in the adjacent tracheobronchial lymph nodes. In the left lung there is latent apical tuberculosis, with caseation and fibrosis. (From the *Journal of Experimental Medicine.*)

under usual conditions in lower animals. Nevertheless, a chronic disease resembling the adult type of pulmonary tuberculosis may be reproduced by inoculating animals which have been rendered resistant by a preceding infection with tubercle bacilli.

Fig. 2.—Diagram of the lungs shown in Fig. 1. (From the *Journal of Experimental Medicine.*)

Pulmonary Tuberculosis of Adults

Pulmonary tuberculosis of adults, in contrast with the childhood type, has its origin in the apex of the lung (Figs. 1 and 2), pursues a chronic course with production of fibrous tissue and when advanced causes the formation of cavities. Especially noteworthy is the absence of tuberculosis in lymph nodes adjacent to the lesion, for even though a whole lobe is affected the lymph nodes at its hilum are not caseous; whereas during childhood, on the contrary, tuberculosis of lymph nodes in most instances progresses more rapidly than the pulmonary lesion from which it arose. In this respect phthisis of adults resembles a second infection produced experimentally.

It is not generally recognized how frequently apical tuberculosis of adults pursues a latent course. In one of five adults who have

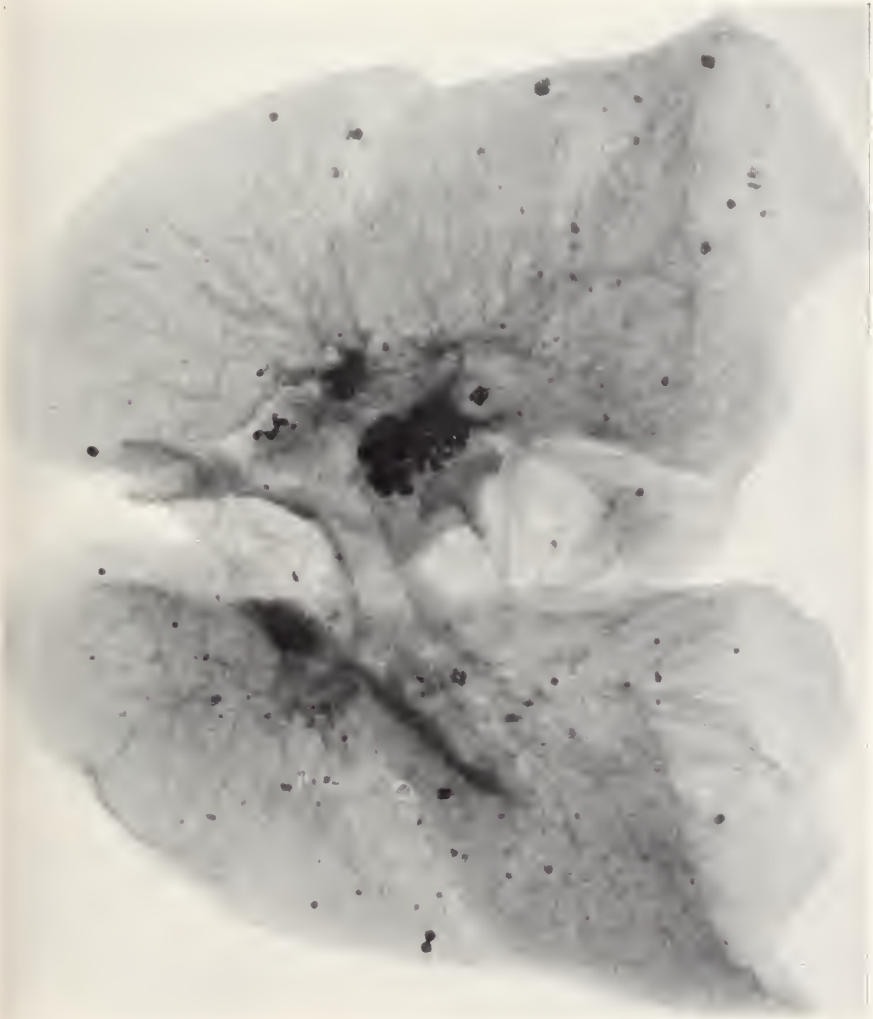


FIG. 3.—Roentgenogram prepared from lungs of a woman, aged thirty-six years, who died following panhysterectomy for myomata. The lungs contained widely scattered calcified nodules and there is massive calcification of the tracheobronchial lymph nodes. (From the *Journal of Experimental Medicine.*)

died from causes other than tuberculosis, there are in the apices fibrocaseous lesions which contain living tubercle bacilli. Occasionally healing is far advanced and caseous spots have undergone calcification. In a considerable number of those who have died from causes other than tuberculosis, there are scars at the apices consisting of fibrous tissue, with none of the gross or microscopic characters of tuberculosis; nevertheless, tubercle bacilli can be recovered from one in four of these scars, which are doubtless, with few if any exceptions, the result of tuberculous infection. All of these lesions have pursued a latent course; they have caused no symptoms evident to the patient and have not been recognized by the physician.

Living Bacilli in Latent Lesions

At the Henry Phipps Institute, Dr. Aronson and I have sought to determine how frequently the latent lesions which have been described contain living tubercle bacilli capable of producing tuberculosis when injected into susceptible animals. In common with many other observers, we have found that material from calcified nodules of the lungs often cause tuberculosis when injected into guinea-pigs. Living tubercle bacilli have been obtained from 20 per cent of 145 nodules examined. From latent fibrocaseous lesions of the apex living tubercle bacilli have been recovered in 76.2 per cent of those examined, and if larger pieces had been used for the tests, it is probable that the microörganism would have been found more frequently. When the apical lesion has completely healed, a fibrous scar with no characters suggesting tuberculosis remains, and from these scars, in 24.4 per cent of those examined, living tubercle bacilli have been cultivated. Since calcified nodules of a first infection are not infrequently found in the same lung with apical lesions, it is possible that tubercle bacilli recovered from calcified nodules have come from the associated apical lesions. We have obtained living tubercle bacilli from calcified nodules removed from lungs with latent apical lesions in 30 per cent of those examined; whereas, when no apical lesions have been present, tubercle bacilli are recoverable in only 8.1 per cent. Living tubercle bacilli disseminated perhaps throughout the organ from the apical lesion may be present in the lung tissue surrounding the calcified nodule.

When tests have been made to determine if lung tissue which

exhibits no evidence of gross or microscopic tuberculosis contains tubercle bacilli, the results are surprising. One-third of pulmonary apices with no gross or microscopic evidence of past or present tuberculosis have contained living tubercle bacilli, and lung tissue from the bases have contained the microörganism only slightly less frequently. It is an old observation, first made by H. P. Loomis, that normal pulmonary lymph nodes may contain tubercle bacilli, and in one-fourth of those examined we have found the microörganism. By examining in a series of autopsies (33), pulmonary apex, base and lymph nodes within or near the lungs, we have found tubercle bacilli in approximately one-half of these autopsies. From our study we have drawn conclusions which are concerned with tuberculous infection: (1) Tubercle bacilli demonstrated by inoculating calcified nodules into susceptible animals, with but few if any exceptions, have not been present in the calcified nodules themselves, but in the lung or lymphoid tissue surrounding them. (2) Since focal lesions of childhood are calcified when apical lesions of adults make their appearance, it may be assumed that the lesion of the adult is not derived from that of the child.

Recognition of Latent Tuberculosis

Recognition of the widespread occurrence of latent tuberculosis in children and in adults gives a broader basis for the investigation of the epidemiology of the disease. Since latent tuberculosis has been defined, solely for the sake of convenience, as that unaccompanied by symptoms evident to the patient or by physical signs recognized by the physician, there are at present only two effective means by which tuberculous infection may be discovered, namely, by the tuberculin reaction and by characteristic changes in *x*-ray plates.

A study of the pathological anatomy of latent tuberculosis which I began about ten years ago has shown that *x*-ray plates prepared from lungs excised after death, afford the most satisfactory means of depicting the size and position of latent tuberculous lesions in the lungs. Comparison of the plates with lesions exposed by dissection has shown how accurately the *x*-ray plate defines both focal lesions of childhood and apical lesions of the adult. Experience has later shown that *x*-ray plates, made from the chest during

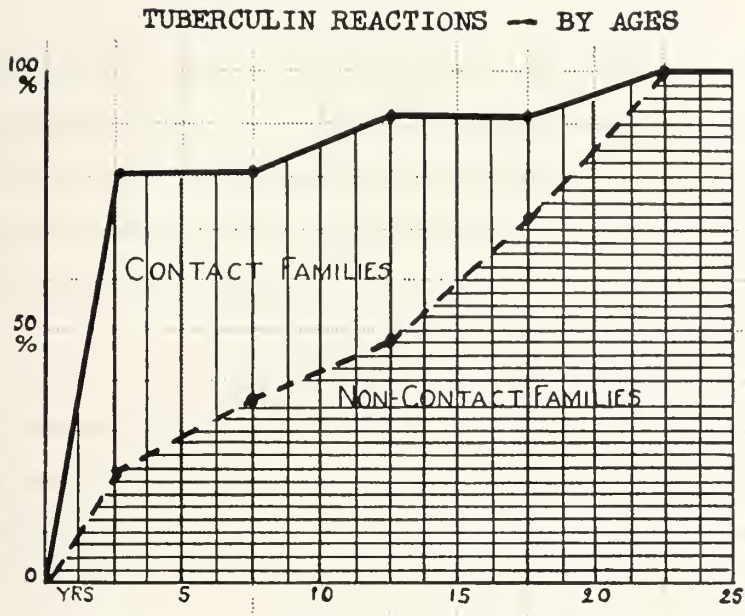
life, reveal lesions identical in form, size and position with those evident in plates prepared from excised lungs.

A study of the spread of latent tuberculosis has been undertaken, at the Henry Phipps Institute, by Dr. McPhedran and myself. By means of the tuberculin reaction and by *x*-ray plates, we have measured the frequency of latent tuberculosis in families classified in relation to their contact with tuberculosis.

In families of which one or more members have suffered with pulmonary tuberculosis and have expelled tubercle bacilli with the sputum, the incidence of latent and manifest tuberculosis has been very high. In another group of families, a member has suffered with tuberculosis, but no tubercle bacilli have been found in the sputum by the usual methods of examination. In these families the incidence of latent tuberculosis, recognized by roentgenological examination, has been fairly high, but in some instances the tuberculin reaction has disappeared. In families with suspected tuberculosis the incidence of latent tuberculosis, measured by *x*-ray and by tuberculin reaction, has not been greater than that in non-contact families.

We have accurately analyzed 100 families under observation during two years, but have based our conclusions upon a much larger number, now approximating 400 families. These have been studied in the dispensary of the Henry Phipps Institute, where we have had the cordial coöperation of Dr. Landis and members of the medical and nursing staff. In families with no tuberculosis, members less than ten years of age are in only small part (30 per cent) sensitive to tuberculin, and on roentgenological examination (Fig. 4) much less frequently (10 per cent) exhibit lesions of latent disease. In families with open tuberculosis, on the contrary, the incidence of the tuberculin reaction is high in younger children (80 per cent) and demonstrable latent lesions occur in almost one-third (30 per cent) of those examined. It is noteworthy that these figures demonstrate the existence of sources of infection outside as well as inside the family, and we are now engaged in a study of the spread of tuberculosis among school children.

Of 124 children from families with open tuberculosis in a parent (Fig. 5), approximately one-fourth have had isolated nodules in the lungs. Another fourth have had massive tuberculosis of tracheobronchial lymph nodes. Four adolescent children have had latent apical tuberculous and 11 children have suffered with manifest



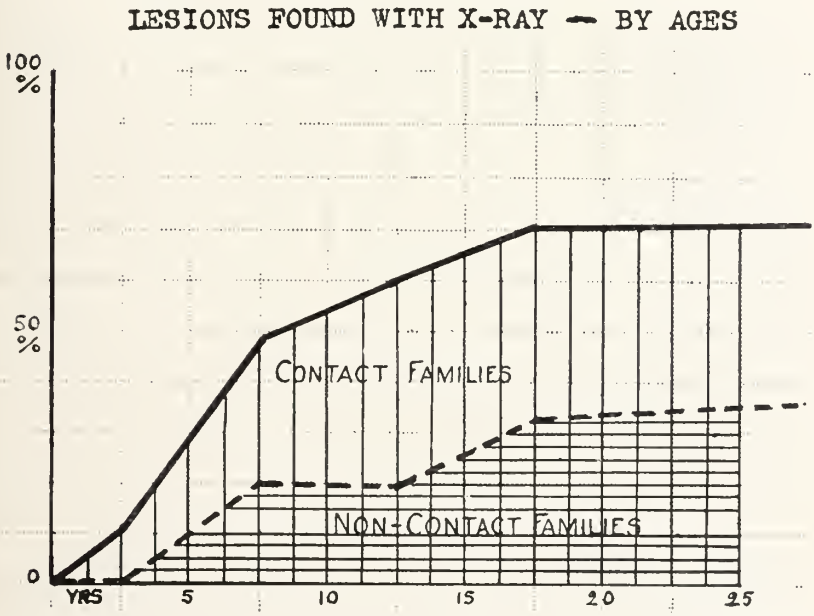


FIG. 4.—Charts showing (1) tuberculin reactions arranged in contact and noncontact families, and (2) lesions found by roentgenological examination arranged by ages in contact and noncontact families. (From the *American Review of Tuberculosis.*)

FAMILIES WITH TUBERCULOSIS IN PARENT (TUB. BAC. IN SPUTUM)									FAMILIES WITH NO HISTORY OF TUBERCULOSIS							
FAMILY	FATHER	MOTHER	NO LESION	PULMONARY NODULE	TUBERCULOSIS TRACHEO-BRONCH. NODES	LATENT APICAL	MANIFEST TUBERCULOSIS		FAMILY	FATHER	MOTHER	NO LESION	PULMONARY NODULE	TUBERCULOSIS TRACHEO-BRONCH NODES	LATENT APICAL	MANIFEST TUBERCULOSIS
Ac	◤	TBC			◉◉		TBC		Ab			XXX				
Bc	TBC	◤	XXXXXX	●●●	◉				Ba			X				
Ca		TBC			◉◉◉◉				Bd			XXXXX				
Ce	TBC		XXX						Be			X				
Cg	TBC	TBC	X						Bf			XX				
Ck	TBC	◤		●●	◉◉				Bh		◤	XXXXXX	●			
Da		TBC	X		◉		TBC		Bi			XXXX				
Df	TBC		X	●					Ci			X				
Dh	TBC	◤	XX		◉				De			XX				
Do		TBC		●●					Dg			XXX	●●			
Ea	TBC		X	●●●●			TBC		Di			XXXX				
Fe	TBC		XXXXX						Dl			XXX	●			
Ia	TBC	◤	X		◉		TBC		Dm				●			
Ja	TBC	◤	XXXX	●●					Dn			X	●	◉		
Je		TBC	XXX	●●					Fa			XXXXX				
Jf		TBC			◉		TBC		Fb			X				
Ka	TBC			●	◉◉	◤	TBC TBC		Fc			XX				
Mb	TBC	TBC	XX						Fg			XXX	●			
Me	TBC		XX	●●●	◉				Ga			X	●			
Mg	TBC				◉◉				Gc			X	●			
Mh	TBC	TBC		●●●	◉◉◉		TBC TBC		Gd			X				
Ml	TBC		XX	●					La				●			
Na	TBC		XX						Mf			X	●	◉		
Oa	TBC		XXX	●●●					Mi			XXX		◉		
Pb	TBC		XXXXXX	●					Mn			XXX				
Sc	TBC		X	●●●	◉				Oc			X				
Sd	TBC		X		◉		TBC		Pa			XXX				
Sf		TBC	X		◉				Sa			XXXX				
Sk		TBC	X	●	◉				Se			XX	●			
Sm		TBC	XXX	●					Sg			X				
Tb	TBC	◤	X	●					Sh			XX				
Vc	TBC		XXXX	●●					Tc				●			
Ya	TBC		XX		◉		TBC		Va			XXXX	●	◉		
Zc	TBC			●●	◉◉	◤										

Fig. 5.—Tuberculous lesions found by roentgenological examination (1) in families of which one parent had pulmonary tuberculosis with tubercle bacilli in sputum, and (2) in families of which no member had tuberculosis. (From the *American Review of Tuberculosis.*)

tuberculosis. In a larger group of families which later came under observation the opportunity to study each of these lesions has been greater.

Isolated nodules of the lung have apparently little significance and, representing a relatively scant infection, are not an evident source of danger to the individual.

Tuberculosis of Tracheobronchial Lymph Nodes

Tracheobronchial lesions conspicuous enough to be mapped out in *x*-ray plates demonstrate the presence of advanced infection. In this group we have included only those lesions which have been sharply defined at the hilum of the lung or in contact with the trachea (Figs. 9 and 11). Accentuation of the hilum shadow or thickening of the mediastinum has not been regarded as evidence of tracheobronchial tuberculosis. The lesion has usually been found in children long exposed to open tuberculosis, the average duration of contact being four years and five months.

Tracheobronchial tuberculosis has doubtless been recognizable in *x*-ray plate in most if not in all instances, because there is some deposit of calcium salts within caseous material, but beginning calcification cannot be regarded as evidence that the lesion has healed. Impregnation with calcium in quantity insufficient to alter the consistence of caseous matter may cast a well-defined shadow upon an *x*-ray plate. Furthermore, it is not infrequently evident at autopsy that a lesion which shows retrogression in one part is active and extending elsewhere. Of greater significance is the occurrence of superimposed infection, acquired as the result of continued exposure to the same source of infection which after several years of contact has caused tracheobronchial disease.

Frequent association of tracheobronchial tuberculosis with manifest disease and the development of clinically recognizable disease in children who already have tracheobronchial tuberculosis has shown that the lesion has grave significance. In some instances pulmonary infiltration accompanied by physical signs has been found in the lung field peripheral to the altered lymph nodes, but more frequently, particularly in adolescent children, it has affected the apex. The affected apex is just as often on the opposite as on the same side with the lymphatic lesion. The evidence of tracheobronchial disease seen in an *x*-ray plate should be regarded as a

sign of grave infection, rather than an accurate record of the lesion itself, for it often fails to define the extent of pulmonary disease. The accompanying tuberculin reaction may give valuable evidence concerning the activity of the process; with an intense reaction a progressive lesion may be feared, whereas with scant or no reaction the disease is probably in process of healing.

Latent Apical Tuberculosis of Adolescent Children

Latent apical tuberculosis of adolescent children (Figs. 9 and 11) is another lesion of grave import, for we have found that it is scarcely separable from apical disease recognizable by physical signs and symptoms. In one small group of families with long exposure to open tuberculosis we have found four instances of latent apical tuberculosis occurring in children in the second decade of life, and later we have seen other similar lesions. In some adolescent children in the group of contact families, widespread infiltration of the apex has been recognized in roentgenograms and has been associated with apical râles, but the patients have been well nourished and apparently in good health. In others there has been the usual evidence of apical disease, namely, cough, loss of weight, cavity formation and tubercle bacilli in the sputum.

It is not possible to say what determines the occurrence of manifest apical disease. In some instances apical tuberculosis of young adults has been evident since adolescence, and in other instances the disease, having its origin in adolescence, has doubtless remained masked during a considerable period of years. Observations of Newsholme indicate that there is often a long latent period between exposure to infection and appearance of phthisis. These considerations furnish evidence that unrecognized pulmonary tuberculosis which has had its origin in adolescence may later manifest itself in early adult life. It is, however, noteworthy that this disease is not the continuation of an infantile type of infection, but has the characters of a second infection acquired during adolescence.

Marital Infection

Our studies of latent tuberculosis have decisively answered the question, Is tuberculosis acquired during adult life? I have already cited the wide difference of opinion concerning the transmission of

tuberculosis from husband or wife to the healthy consort. Here, again, definite figures are obtainable from the small group of families which, having a member with open tuberculosis, have been under observation during two years, but similar data are evidently obtainable from a much larger group of families which have later come under observation (Fig. 5). In three families both parents have suffered with tuberculosis recognizable by the usual diagnostic methods. In seven instances in which one consort has suffered with open tuberculosis, roentgenological examination has demonstrated the presence of a latent apical lesion in the other. These lesions have reproduced the picture of latent apical disease seen in x-ray films prepared from excised lungs of those who have died from causes other than tuberculosis. Shadows are seen at the margin of the second rib, and salients project downward into the lung substance. Occasionally, conspicuous rounded shadows occur in the second and third interspaces at the outer aspect of the lung. In 11 instances, examination of the apparently healthy consorts of those with open tuberculosis have revealed no apical lesion, so that of 21 instances in which both parents have been examined there has been demonstrable tuberculosis in 10, that is, in approximately one-half. The incidence of latent apical disease in husband and wife of families with no history of contact with tuberculosis has been only 1 in 13.

When latent tuberculosis is taken into consideration, more accurate insight into marital contagion is obtained. Our study, even though the number of individuals concerned is small, shows that the healthy consort of husband or wife suffering with open tuberculosis may become infected. It is noteworthy that a long period of contact is necessary, and the average period during which the infected partner has been in contact with open tuberculosis has been seven years and eleven months.

The difficulty of collecting accurate statistics concerning the appearance of manifest tuberculosis resulting from marital infection is obviously great, for the disease may remain latent during a long period. Since these latent apical lesions, as we have found, contain living tubercle bacilli even when they have been converted into fibrous scars, the possibility of transformation of latent into progressive disease is by no means negligible. Individuals in whom latent apical tuberculosis has been recognized should be treated as though they had recovered from tuberculosis.

What is regarded as incipient tuberculosis is, in many and doubtless in most instances, latent disease which has recently become manifest. What are the conditions which transform latent into active disease is not clearly understood; but if the existence of latent tuberculosis is recognized the patient can adopt, under advice of his physician, a course of life which will tend to prevent the occurrence of progressive disease.

Infected Family as a Unit

Our study has emphasized the importance of regarding the infected household as a unit. The occurrence of latent tuberculous lesions, recognizable by *x*-ray plates or by tuberculin reaction, in members of a family is an index of their exposure to tuberculosis.

EXPLANATION OF FIGURES 6 TO 11.

Family charts, Figs. 6 to 11, depict the distribution of tuberculosis in families, and diagrams show important lesions found in roentgenograms of members of these families. Brief clinical notes explain the progress of the disease in each family.

Vertical lines represent each member of the family and correspond in length with the duration of residence within the household. They are erected upon a horizontal line representing the date at which the family came under observation, and in most instances show the duration of married life of parents and the ages of children. Whenever a member of a family leaves the household the corresponding line is broken; for example, by residence in a sanatorium. Observations made after the original date of the chart are recorded below the horizontal line representing this date.

Tuberculin reactions are indicated by 0 (negative), or from 1 to 3 + signs over the quantity in milligrams of tuberculin used. Symbols are used to indicate tuberculous lesions: A heavy black dot at the left of the life-line represents a focal tuberculous lesion of the lung; an encircled dot is tracheobronchial tuberculosis; an arc with a salient projecting downward is latent apical tuberculosis. A cross upon the life-line indicates that roentgenological examination has been made at the date indicated with negative results. A heavy black column at the right of the life-line indicates manifest tuberculosis; an additional column on the left indicates that the sputum contained tubercle bacilli. The result of sputum examination (Sp) is indicated by 0 or + placed above the number of examinations. A series of dots on the right of the life-line indicates that tuberculosis has been suspected. (The figures with the accompanying explanatory notes are from the article of Opie and McPhedran, "The Contagion of Tuberculosis," *American Review of Tuberculosis*, 1926, xiv, 347.)

Fig. 6.—Family Gd. There is no known contact with tuberculosis. Ida has mitral regurgitation and has chorea. *Housing*, six rooms and bath for six persons; *cleanliness*, fair; *diet*, sufficient; *financial status*, fair.

Fig. 7.—Family Sa. The father died of influenza in 1918. There is no known contact with tuberculosis. *Housing*, four rooms for seven persons; *cleanliness*, fair; *diet*, sufficient in quantity but low in protein; *financial status*, poor.

Remarks. The only evidence of tuberculous infection is a positive tuberculin reaction in the oldest child and this after approximately two years has diminished in intensity.

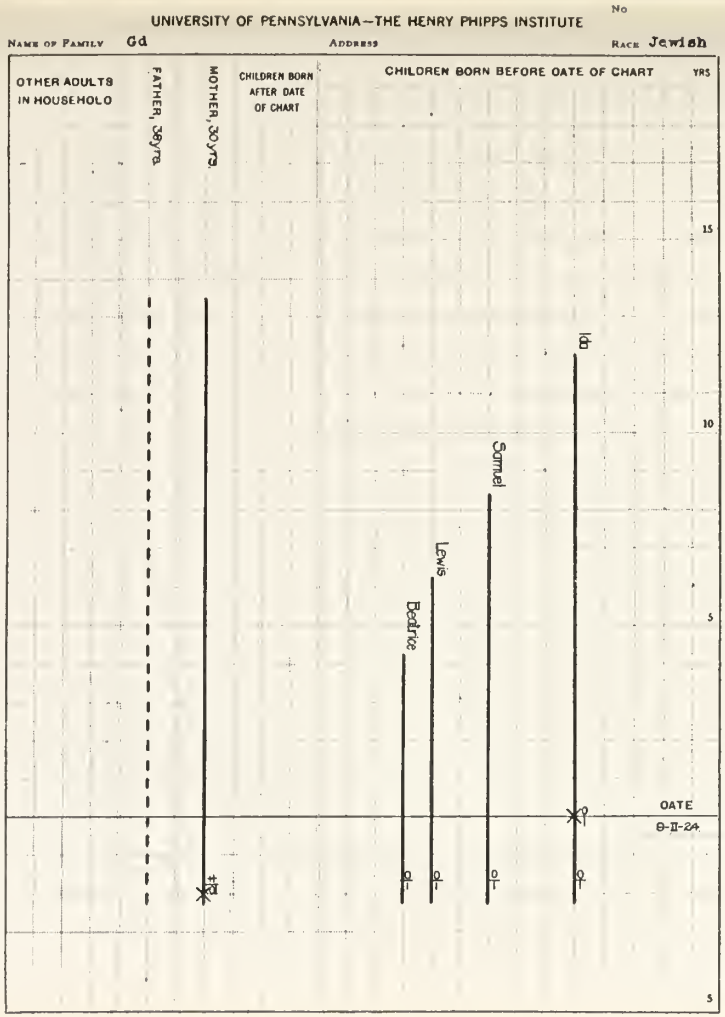

UNIVERSITY OF PENNSYLVANIA—THE HENRY PHIPPS INSTITUTE
No
Name of Family Gd
Address
Race Jewish
OTHER ADULTS IN HOUSEHOLD
FATHER, 36yrs
MOTHER, 30yrs
CHILDREN BORN AFTER DATE OF CHART
CHILDREN BORN BEFORE DATE OF CHART
YRS
15
10
5
5
Ida
Samuel
Lewis
Beatrice
DATE
9-II-24

Fig. 6

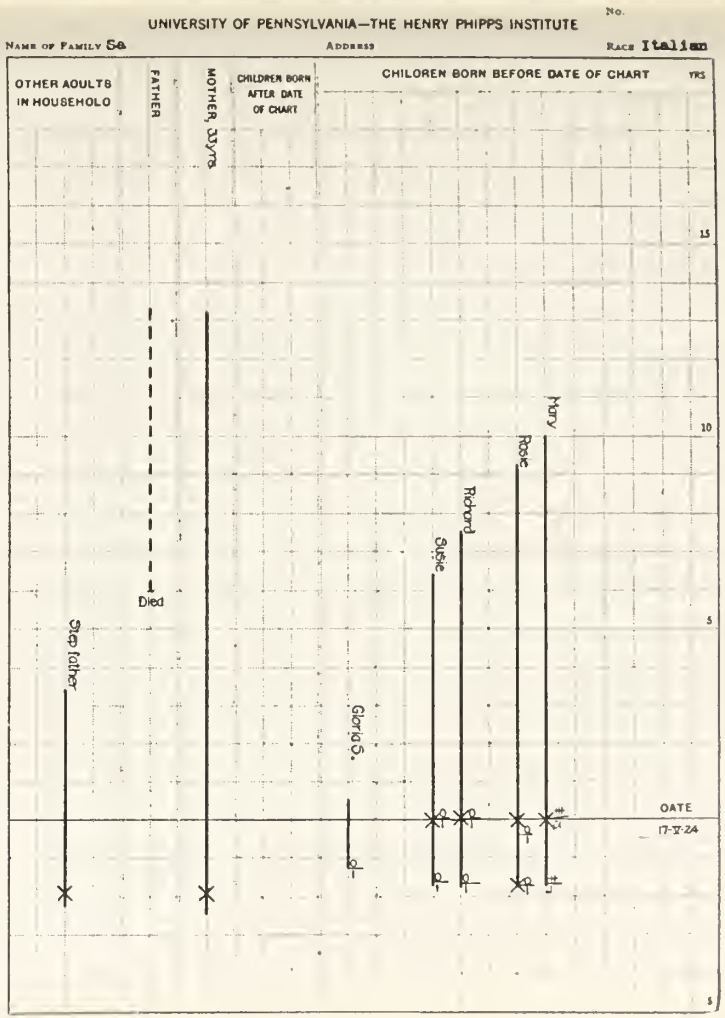

UNIVERSITY OF PENNSYLVANIA—THE HENRY PHIPPS INSTITUTE
No.
Name of Family Sa
Address
Race Italian
OTHER ADULTS IN HOUSEHOLD
FATHER
MOTHER, 33yrs
CHILDREN BORN AFTER DATE OF CHART
CHILDREN BORN BEFORE DATE OF CHART
YRS
15
10
5
5
Mary
Rose
Richard
Susie
Gloria S.
Died
Step father
DATE
17-X-24

Fig. 7

In varying degree it suggests existing contact and may furnish evidence of a past infection, even though the individual who has been its source has recovered from his disease or is no longer a member of the household. The activity of the tuberculin reaction, especially in the younger members of a family, measures the gravity of existing contact with open tuberculosis, for there is abundant evidence that the tuberculin reaction diminishes in activity and finally disappears when the tuberculous lesion which produces it has healed.

Furthermore, the presence of latent tuberculosis, demonstrable by tuberculin reaction or by *x*-ray plates, in a group of individuals living together indicates contact with tuberculosis, and when one member is suspected may confirm a diagnosis incompletely established by the usual clinical methods. We have repeatedly sought for tuberculosis in the parent because latent infection has been widespread in the children and have succeeded in confirming our suspicion. The parent has unwittingly made his children a diagnostic sign of his own disease.

FIGS. 8 AND 9.—FAMILY AC. *Probable source of contact:* (1) Mother; (2) maternal aunt. *Duration of contact:* (1) Mother, seven years, ending within the period of observation; (2) maternal aunt, four years, still continuing.

Pulmonary tuberculosis of the mother. Cough and expectoration had been persistent for six months. The physical signs were those of a far-advanced lesion. She was said to be careful after diagnosis in disposal of sputum.

Latent apical lesion of the father. Joseph Ac reports no complaints and is very well nourished. Temperature and pulse are normal. Roentgenograms of February, 1925, and 1926 show moderately dense infiltration of the right apex and slighter infiltration of the right apex.

Pulmonary tuberculosis and tuberculosis of tracheobronchial lymph nodes of the maternal aunt. Celia M., in February, 1922, complained of slight cough with scant white expectoration. The sputum has been occasionally streaked with blood. The patient had gained 20 pounds in February, 1926. Temperature and pulse were always normal. Roentgenograms of February, 1925, show a granular mass (calcified), 1.4 x 0.8 cm., at the left bronchial bifurcation, and a nodule 0.5 cm. peripherally in the lung. There is moderately dense infiltration of the left apex and a slight infiltration of the right apex.

Pulmonary tuberculosis and tuberculosis of tracheobronchial lymph nodes in a sister. Anna, aged twelve years, was first seen, November, 1923. Roentgenograms showed a sharply defined nodule (calcified) near the base of the right lung, and three granular masses, one in the hilum of the right lung, one at the bifurcation of the trachea and one to the right of the trachea, measuring 1.0 x 0.6 cm., 1.7 x 1.2 cm. and 1.0 x 0.8 cm., respectively. Râles were heard at this time over the left apex posteriorly.

Tuberculosis of tracheobronchial lymph nodes in a sister. Rose was first seen, November, 1923. Roentgenograms showed granular masses (calcified), one in the right hilum, 0.6 x 0.3 cm.; and one below the carina, 1.7 x 0.8 cm. *Housing*, four rooms and bath for five persons; *diet*, sufficient; *cleanliness*, good; *financial status*, fair.

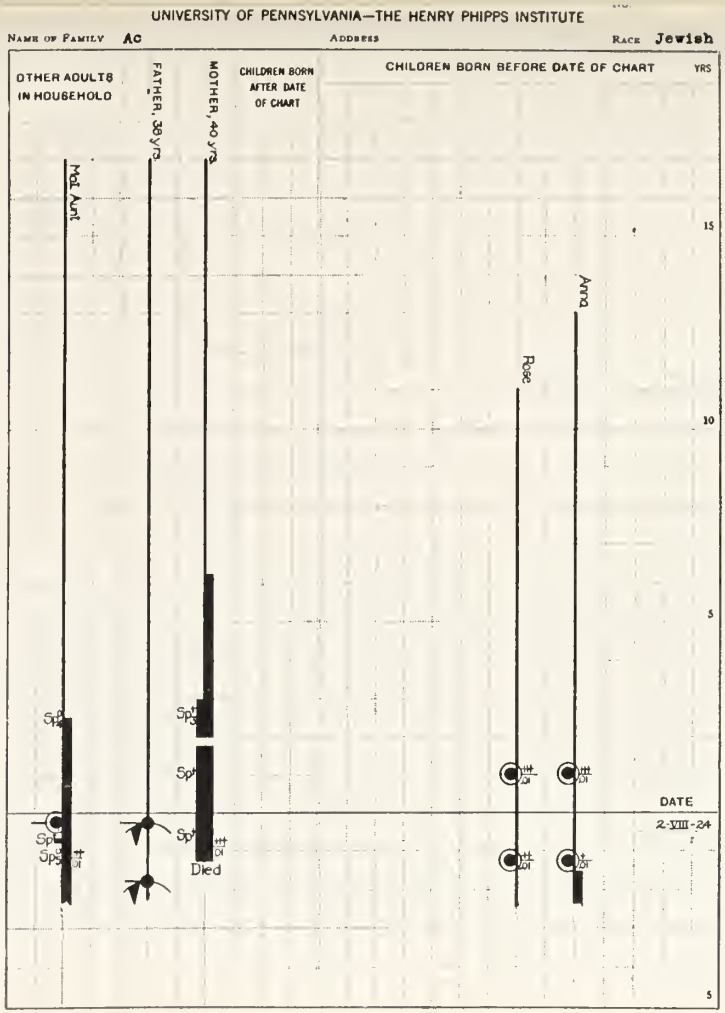

UNIVERSITY OF PENNSYLVANIA—THE HENRY PHIPPS INSTITUTE
NAME OF FAMILY Ac
ADDRESS
RACE Jewish
OTHER ADULTS IN HOUSEHOLD
FATHER, 38 yrs
MOTHER, 40 yrs
CHILDREN BORN AFTER DATE OF CHART
CHILDREN BORN BEFORE DATE OF CHART
YRS
Mat. Aunt
Rose
Anna
Died
DATE
2-VIII-24

Fig. 8

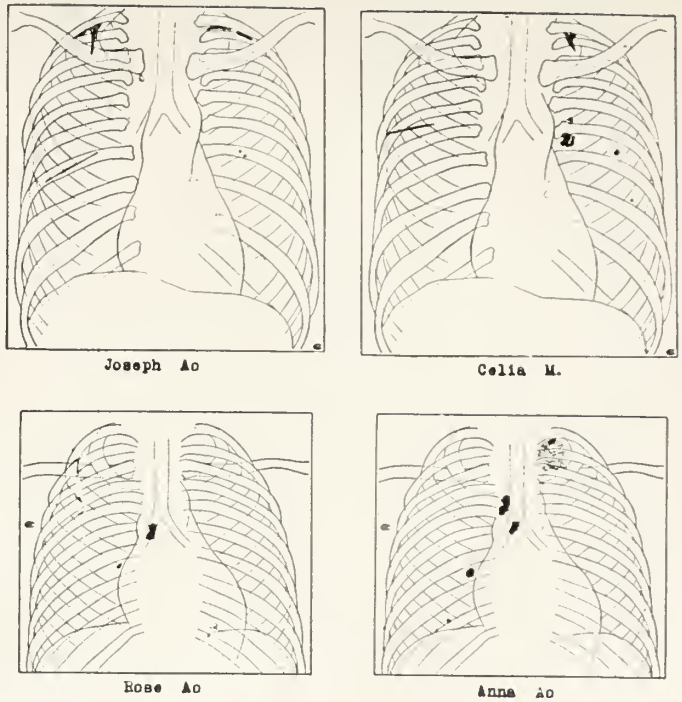

Joseph Ac
Celia M.
Rose Ac
Anna Ac

Fig. 9

By the methodical examination of families it may be shown that households exposed during many years to contact with open tuberculosis have become, as it were, saturated with the disease. (Figs. 8, 9, 10, 11). Every member has an intense tuberculin reaction, the younger children exhibit tracheobronchial tuberculosis and one or more of the older children suffer with manifest disease. It is not necessary, as heretofore, to wait the development of manifest disease in order to determine how the family has fared under existing exposure to infection, since adequate tests will reveal threatened danger.

When such families live in poverty, crowded into a room or two and with inadequate food, municipal authorities might well consider the economy of placing them under conditions which will prevent the development of further disease, for the ultimate cost of combatting disease, hopeless when it is finally recognized, will doubtless be far greater. When existing latent and manifest disease is depicted by the graphs (Figs. 6, 7, 8, 10) which we have employed, the imminence of danger is evident.

It is often assumed that children infected with tuberculosis are immune from subsequent infection. Evidence from experimental animals and from clinical observation indicates that a first infection confers no immunity which cannot be overcome by massive infection. Our observations indicate that tracheobronchial tuberculosis which has occurred after several years of contact with open tuberculosis

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FIGS. 10 AND 11.—FAMILY ZC. *Source of infection*, father. *Duration of contact*, four and and a half years, ceasing within the period of our observation.

Pulmonary tuberculosis of the father. He was first seen, June, 1920, with far-advanced pulmonary tuberculosis and tuberculous laryngitis. He was careless in disposal of his sputum.

Latent apical tuberculosis of sister. Mary had no symptoms and her weight was normal. Roentgenograms showed a left apical infiltration. She was married recently and is apparently well.

Tuberculosis of tracheobronchial lymph nodes. (1) Roentgenograms of William in January, 1926, show a sharply defined nodule (calcified) in the right lung anteriorly and three granular masses (calcified), one, 1.8 x 0.8 cm., close to the right bronchus; one, 2.1 x 1.3 cm., at the carina, and one, 2.1 x 0.7 cm., to the right of the trachea; he was free from symptoms and his weight was normal. (2) Helen's roentgenograms of January, 1926, show four granular masses (calcified), one on the right bronchus, 0.7 x 0.5 cm.; one on the left bronchus, 0.6 x 0.4 cm.; one near the carina, 1.2 x 0.7 cm.; one to the right of the trachea, 1.8 x 0.6 cm. There is a soft shadow in the left base. She had no symptoms.

Housing, two rooms for seven persons; *cleanliness*, poor; *diet*, poor in protein, mostly starches; *financial status*, poor.

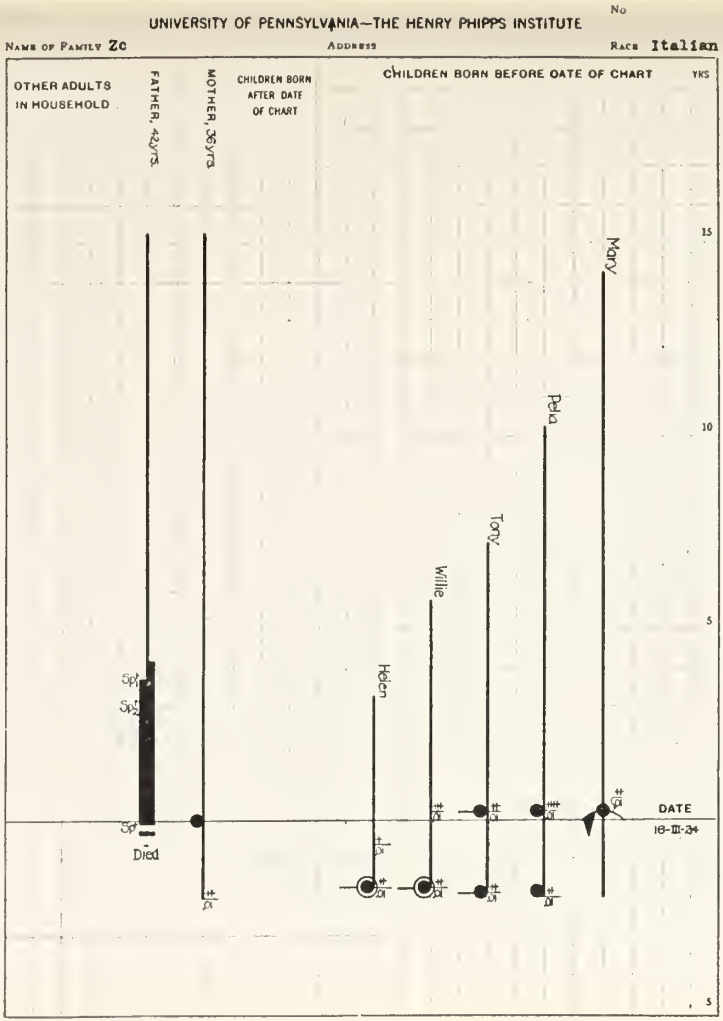
UNIVERSITY OF PENNSYLVANIA—THE HENRY PHIPPS INSTITUTE
No
Name of Family Zo
Address
Race Italian
OTHER ADULTS IN HOUSEHOLD
FATHER, 42yrs.
MOTHER, 36yrs.
CHILDREN BORN AFTER DATE OF CHART
CHILDREN BORN BEFORE DATE OF CHART
YRS
15
10
5
5
Mary
Pelia
Tony
Willie
Helen
Sp
Sp
Sp
Died
DATE
18-III-24

Fig. 10

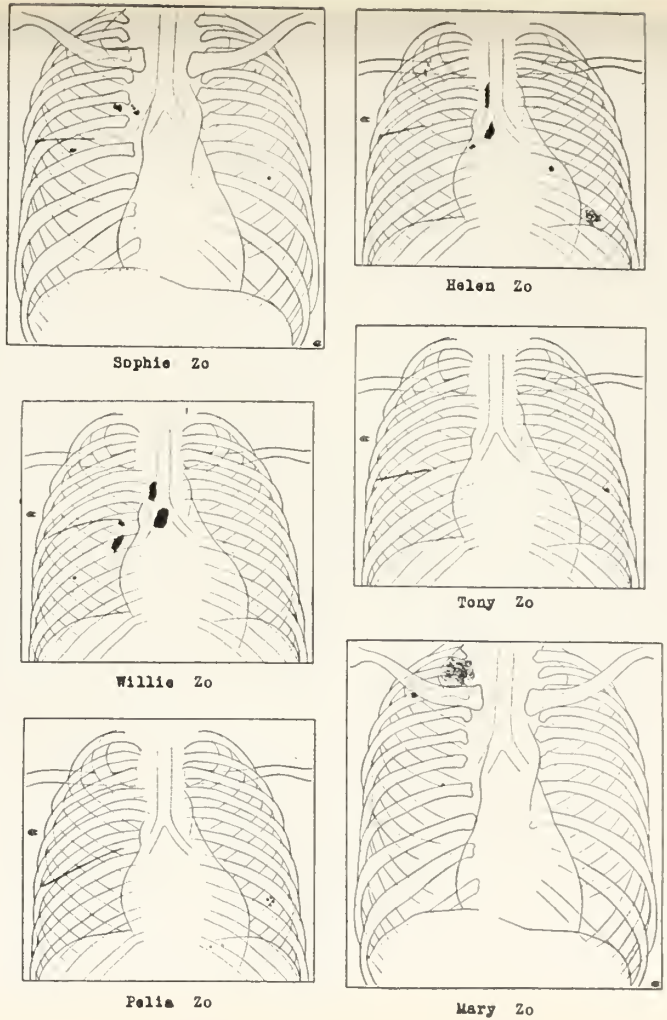
Sophie Zo
Helen Zo
Tony Zo
Willie Zo
Pelia Zo
Mary Zo

Fig. 11

may be followed by apical disease which is presumably the result of continued exposure to the same source of infection. In lungs of adolescents and of adults examined after death, focal pulmonary nodules and extensive tracheobronchial tuberculosis are often found in association with apical tuberculosis, but there is no indication by continuity that apical disease has developed from the earlier lesions.

Care of Families Exposed to Open Tuberculosis

The routine methods for finding tubercle bacilli in the sputum serve to identify those individuals who are responsible for the spread of tuberculosis within families. The chief factors in the campaign to combat tuberculosis are official notification, social-service care and education of the tuberculous and isolation in hospitals of those suffering with advanced disease. It is obvious that no method for the diagnosis of tuberculosis should be neglected, but multiple sputum examinations furnish the most decisive means for finding those who are a danger to their neighbors. The value of a dispensary in preventing tuberculosis may be measured by the frequency with which the sputum of its patients is examined. If prophylactic care of families is guided by knowledge of existing danger, the efficiency of social service may be increased with no increased expenditure. One or two examinations of the sputum are not sufficient, but they must be repeated at frequent intervals as long as the patient is under treatment.

All members of families which have been exposed to open tuberculosis should be subjected to repeated examination, so that latent lesions which threaten manifest disease may be detected as soon as they are recognizable. During the first two years of life, children of tuberculous parents should receive intradermic tuberculin tests at intervals of six months and subsequently at yearly intervals. A positive tuberculin reaction in an infant suggests grave infection, but recovery may doubtless occur. The significance of the reaction diminishes with increasing age, but a severe reaction (with 0.01 cc. of tuberculin) indicates the presence of active infection. Roentgenograms seldom reveal tuberculous lesions in their earliest stage, but the recognition of extensive tracheobronchial disease in the presence of an active tuberculin reaction has grave significance, for it may be a forerunner of manifest tuberculosis. Roentgenological

examination repeated at intervals may disclose during the second decade of life, and more frequently during adolescence, the presence of latent apical lesions which by insensible stages pass into clinically recognizable disease. Furthermore, it is desirable to subject to similar examination adult members of households exposed to open tuberculosis, for our study has shown the frequency with which latent apical tuberculosis makes its appearance in consorts of tuberculous individuals. Recognition of the latent lesions which have been enumerated will suggest means by which subsequent disease can be prevented, for it may be assumed that if well-known measures which retard the progress of manifest tuberculosis are employed, the transformation of latent into active disease will be prevented.

Conclusion

Though it has long been known that tuberculosis is an endemic disease which, like measles today and smallpox two centuries ago, is so widely distributed that few escape, little importance has been attached to latent infection. Many seem to have thought that the isolated tuberculous foci well known to pathologists have little in common with the disease recognizable by clinical methods. There is no line of separation between the two. Tuberculosis, which is nearly universal in the crowded populations of today, is like a submerged mountain range with volcanic peaks projecting above the surface. We forget the widespread infection which is out of sight and fix our attention upon the visible disease.

Latent tuberculosis, doubtless, increases resistance and often affords complete protection from subsequent infection; but at the same time it is a source of danger and, neglected, may be transformed into progressive disease.

Latent tuberculosis of childhood confers immunity against subsequent infection, but resistance is limited and transient. Pulmonary tuberculosis of adults is not derived from the disease of childhood, but is the result of new infection and pursues a chronic course, because some immunity induced by preceding disease still persists.

THE POLYMORPHONUCLEAR NEUTROPHILES IN THE BLOOD OF LYMPHADENOPATHIES AND THEIR RELATION TO CLASSIFICATION AND RADIATION TREATMENT*

By HERBERT FOX, M.D.

(From the Wm. Pepper Laboratory of Clinical Medicine.)

The observations presented in this article are intended to show that the neutrophiles may exhibit percentage and actual figures in diseases of the lymphatic glands that are of value in diagnosis, and that the behavior of these cells under radiation treatment may be useful in the control of the exposure to *x*-ray. In addition, the data may assist in the classification of the entire obscure group of the leukemias, lymphatic tumors and lymphogranuloma.

There is an increase of cases presenting morbid lesions in the lymphatic structures, judging by the number of examples seen in large clinics and radiological institutes, or, if the incidence be no greater, the number seems to grow because our honest knowledge of cause, classification, diagnosis and treatment remains the same. The lack of increased understanding is due largely to the indefiniteness of some of the varieties, somewhat to the chronicity of the diseases, but in no small part is it to be ascribed to the fact that we have a method of treatment that gives symptomatic relief for long periods. Ever since Senn demonstrated the value of *x*-ray in the handling of leukemia, it has been the fashion to pass the buck to the radiologist, who proceeds to make life more comfortable for the patient, and incidentally to relieve the conscience of the clinician. But the radiologist is honest enough to admit that, beyond gaining a knowledge of how radiant energy affects gross morbid lesions and reduces circulating leukocytes, he has added nothing to our knowledge of the essence of these diseases. The work of Minot and of Schinz indicates very clearly that all that can be expected from

* Read March 2, 1927.

radiation treatment is comfort, reduction of complications and possibly a slightly increased expectation of life.

Because of the known low resistance of lymphatic leukemia to infectious disease, of which fact we had two striking examples, and because we feel that we hastened, fortunately for us, the inevitable end of a case of lymphadenosis, our attention was drawn to the neutrophiles of the blood as possible indicators of the resistance or lack thereof in these cases. The results have been instructive insofar as individual cases were concerned and they may be of significance in classification and in controlling radiation treatment.

The polymorphonuclear neutrophile reaction is our best indication of response to infection and of the activity of the bone-marrow in this direction. The numerical variations of neutrophiles do not run parallel with those of hemoglobin or red blood cells, for in certain infectious states the first may rise, the others fall. Contrariwise, as we shall see, in certain leukemias, neutrophiles may fall and the red cells and their coloring matter may remain high. This would indicate that the apparatus involved in the production of neutrophiles and of erythrocytes are independent. So, too, the production and destruction of neutrophiles and of lymphocytes are, in the adult at least, unrelated. Nevertheless, the behavior of polymorphonuclear cells is different in different forms of lymphadenopathy and their behavior with radiation treatment is not the same in all varieties.

Before proceeding with the details of cases forming the basis of this report it will be well to discuss briefly, first, the classification used and the methods employed to reach the diagnoses, and, second, the accepted known effects of radiation upon the cells and tissues involved in these diseases.

One may find almost as many classifications as there are serious students of lymphadenopathies, which condition is due to the bewildering variety of clinical and pathological manifestations. There seems to be a gradation, in the sense of morbid anatomy at least, from chronic lymphatic hyperplasias of simple benign character, possibly in response to an infectious virus, to the unrestrained malignant proliferation illustrated by lymphosarcoma. This view would imply the probability of identity in the causative agency of many, if not all, chronic lymphatic hyperplasias. Such may be the case, but we are far from such a decision. Nägeli accepts this view with reservations, because of apparent transitions of one well-

defined state of certain cases into another equally definite pathological and clinical type; for example, a local lymphosarcoma becoming leukemic, a simple lymphadenoma resulting in sarcoma. The present proposal for classification by the Lymph Gland Registry admits benign, borderline and malignant lymphatic hyperplasias, but they are as yet based entirely on microscopic appearances. Sternberg differs from all this viewpoint, and believes that there are clinical and pathological characters that we should be able to identify, and objects to the idea of transition stages although admitting the extence of borderline cases. Ewing's position seems to be that we should attempt to specify types in order to learn more about each one. In our clinic the classification of the serious varieties that demand radiation treatment has been as simple as we could make it, with, at the same time, the attempt to distinguish the main types, admitting that many cases partake of the characters of related forms. There will be given here only the criteria relied upon in judging the diagnoses of the cases used in this article, since the reaction of the neutrophiles is the principal subject of the study.

Chronic lymphatic leukemia is characterized by disproportionate lymphocytosis, moderate lymphadenopathy, moderate or absent splenomegaly, chronicity and favorable action to radiation.

Sublymphemic leukemia is similar, but the number of circulating leukocytes is only moderately increased, with, however, absolute and relative lymphocytosis. Enlarged lymph nodes are more prominent in this condition.

Aleukemic lymphadenosis is generalized lymphadenopathy, with no leukocytosis and no lymphocytosis and no splenomegaly or very slight increase of this organ. The pathology is not so uniformly distributed as in leukemia proper, the lesions often being irregularly scattered and with definitely less tendency to involve the liver, kidney and spleen. It is claimed (Nägeli and others) that this condition may be one phase of leukemia, but it is credited by some writers (Ewing and Sternberg) with being a fairly well-defined entity. Our results would favor the latter idea. Indeed, it approaches lymphosarcoma rather than leukemia. Certain cases end in marrow lesions and bone tumors akin to myeloma. All cases of chronic lymphadenopathy, especially when suffering vague pains, should be studied for osseous lesions. Very little is on record of bony changes in lymphatic leukemia.

Leukosarcoma is a local lymphatic tumor with leukemia, of rapid

course, with a tendency to involve in the late stages the same tissues—spleen, liver, kidneys, marrow—as are affected in leukemia. Its micro-anatomy is identical with leukemia and I have called it "local leukemia." It does poorly under *x*-ray.

Lymphosarcoma is a local tumor of varying rate of growth tending to involve all adjacent tissue (heteroplastic, Sternberg maintains) without leukemia. Nägeli states the lymphocytes are low. Its dissemination is to places not especially favored by leukemia. It does well under *x*-ray treatment. Microscopically the lymphoid cells of lymphosarcoma are separated and supported by a fine fibrous tissue stroma that seems to grow with the tumor. In leukemia, and in the one leukosarcoma that I have studied, no interstitial framework exists beyond that normal to the tissue invaded.

Hodgkin's disease is a widespread growth in lymphatic tissues, usually with local manifestations at the beginning but very shortly becoming general, attended by polynucleosis, an approximately normal number of lymphocytes, early anemia and chronic course. The cellular stage or variety does well with radiation treatment, the fibrotic, poorly. Certain cases assume a rapid phase with the appearance of histology like round- or spindle-cell sarcomata.

Endothelioma may affect gland groups or be generalized in the lymphatic glands. The clinical picture is varied. So far as known there is no typical blood picture.

All varieties of sarcoma may occur in the lymph nodes, springing from reticulum or supporting tissue. There is no typical blood picture; polynucleosis is frequent.

The diagnoses in the cases reported herewith were made by clinical history supplemented by bioscopic adenectomy in every case. A few have been subjected to autopsy with partial or complete confirmation, a few are untraced and the remainder live.

Bone-marrow involvement in lymphatic hyperplasias is not a pathological feature of great frequency, as judged by early and pronounced anemia, although the platelets may be decreased and coagulation retarded. Despite this, neutrophiles, products of the bone-marrow, are low in number in lymphatic leukemia, a condition that suggests an action of the virus or agency that depresses the activity of the marrow. In late stages of leukemia, deposits and infiltrates of lymphoid cells are combined with marrow atrophy or aplasia, mixed with abortive attempts at hyperplasia to make enough blood cells. In the so-called pseudoleukemic states the

marrow and bone itself may be involved by tumorlike nodules of lymphoid cells, or, in the case of Hodgkin's disease, of characteristic histology. This feature will assist in the distinction of leukemia and its immediate relatives (leukosarcoma, sublymphemic or aleukemic leukemia) from lymphadenosis and sarcoma. This will be emphasized at a later time. Involvement of the marrow by secondary neoplasms usually results in a circulating polynucleosis.

The involvement of the marrow and its myeloid series of cells and the behavior of these elements under radiation in relation to lymphadenopathies, deserves for its understanding a brief review of what radiant energy does to the neutrophiles and to the important cellular units of the diseases in question, the lymphocytes. In so far as the kind of cells produced under normal and leukemic conditions are concerned, there does not seem to be evidence that the cells differ in a biological sense. Their capability of function as phagocytes and enzyme carriers is essentially the same. Hektoen seems to think that the neutrophiles of lymphatic leukemia are perhaps above normal in phagocytic power, while in myelemia they are rather lower. The evidence in the literature, and very scant it is, that there is any phagocytic variation in normal and radiated neutrophiles is contradictory. The inflammatory exudates after radiation consist of the same types of cells as when no radiation has occurred.

The effect of radiation upon lymphoid cells was first completely described by Heineke. He showed the destruction of small mononuclears in circulation and in lymph follicles, the fragmentation of lymphoblasts and the phagocytosis of their remains, the locality being replaced by fibrous tissue. The destruction occurs only with certain doses and begins at once. Very small doses are followed by lymphocyte increase. The myeloid cells are affected only by heavier radiation and the changes take place only after some time. This can be observed clinically when, after heavy doses, some days elapse before there is a drop in neutrophiles. Later observations by Aubertin, Schinz, Mottran and others indicate a destruction of myelocytes of the neutrophilic series, but no material change in the eosinophilic and basophilic series. Schisler and Brown observed a relative rise in the number of neutrophiles when myelocytes are destroyed. They were studying myeloid leukemia and found the total number of neutrophiles was reduced more than the percentage. Aubertin's work indicates that regeneration of neutrophiles is manifested by the appearance of young cells, as indicated by the Arneth

formula. He showed, however, that all marrow cells were less sensitive than splenic cells, and he and others have shown that the lymphatic tissues feel the effect of *x*-ray sooner than do marrow tissues, but the effect passes off sooner. In general, it may be said that under pathological conditions, the cell to feel the effect first is the young stage of the variety that is abnormally active. Radium has more effect on the marrow than does *x*-ray, but the nature of the effect is essentially the same, a reduction of myelocytes and of polymorphonuclears, but with the latter relatively more numerous.

The effect of radiation on the marrow is further shown by the low percentage of neutrophiles in roentgenologists and the aplastic anemia that is sometimes observed in them.

There are very few direct references in literature to the changes of lymphatic cells in myeloid leukemia or of neutrophiles in lymphatic leukemia. They tend to show a reduction of the number of lymphocytes in myeloid leukemia without disturbance of their percentage. Stengel and Pancoast, in 1912, warned against treating cases that showed a rapid and very marked drop in circulating leukocytes. Soiland also discusses this, and he and others note the danger of driving down the leukocytes to figures below normal. The best practice seems to dictate stopping the treatment just before normal figures are reached. When in myeloid leukemia there is a relative rise of myelocytes with a fall of total leukocytes, a fatal outcome may be expected. This suggests that the effect of radiation on acute leukemia is dangerous to the extent that young cells are forced to degenerate.

It is, therefore, doubtless that radiation can reduce the production of the leukocytes of the two principal varieties. I have tried to show in our cases that the large monuclears were altered in some definite way, but without success. The eosinophiles tend to rise in some and fall in others, but no regularity could be discovered.

Despite the control by radiation of cellular overproduction and the clinical improvement in most cases by such treatment, there is no evidence that the formative cells are made to behave themselves and to produce the normal cells in normal numbers. Nägeli, in his textbook, writes of an improvement of cytogenesis without explaining its operation; this is open to question. On the contrary, certain cases of all varieties of leukemia tend to reach a resistant stage where no favorable change can be effected. Still again, radiation can be shown to drive down useful cells and at present no selective method is at hand.

We may now pass to a discussion of the cases upon which the present study is based. The figures for the blood counts are illustrated in graphs, the plan of which demands some elucidation. The curves are based upon the blood counts as made under clinical con-

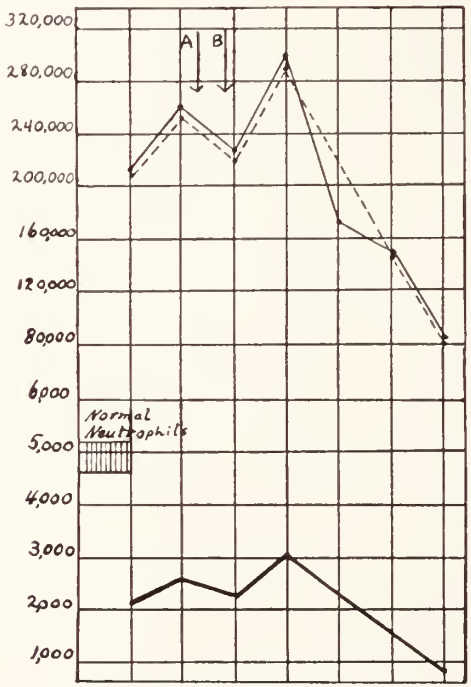

CHART I.—Case I. An example of typical chronic lymphatic leukemia, clinically and from the blood picture, nine weeks' observation; the patient had a decided secondary anemia; the participation of the polynuclear cells in the inactivity of the bone-marrow may be noted; during the course of radiation treatment the neutrophils tended to fall, their number following the course of the total and lymphocyte counts; transfusion was necessary; at all times the neutrophils were below the normal number; *A* indicates transfusion; *B*, roentgen ray begun. In this and the following charts, the heavy lines indicate neutrophils; the dash lines, lymphocytes; the light lines, total leukocytes, and the black squares, death.

ditions. The intervals are not regular, nor are they the same in all the cases. Nevertheless, the course of blood pictures can be followed, and, accepting the diagnoses as correctly made upon the criteria given before, these graphs portray rather definite features of the neutrophile number and reaction during treatment. They

can also be read to support the lessons that are conservatively drawn from them.

The curves are made upon the number, *not the percentage*, of circulating leukocytes, lymphocytes and neutrophiles. At the lower left of the graphs in appropriately shaded spaces are shown the normal limits of each of the three groups. (A calculation of the

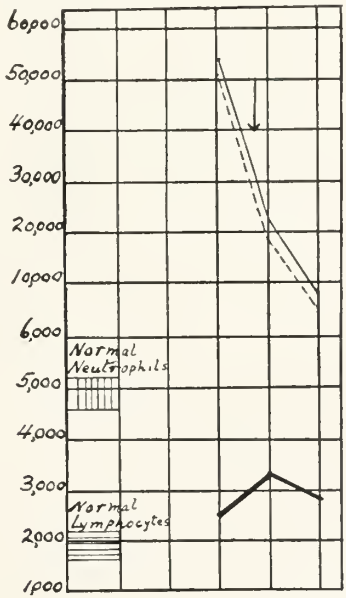

CHART II.—Case II. A case of chronic lymphatic leukemia in which radiation gave fairly good results; one week's observation; the records show only the proportionately low number of neutrophils; the patient reacted well but has had a return and recently had another series of roentgen-ray treatments which are not included; the neutrophils remain low; the arrow indicates roentgen-ray treatment.

exact number of cells should be made oftener in all kinds of cases. When the total is abnormal, one usually fails to realize the exact number of each of the varieties.) The figures given (7000 to 8000 total leukocytes; 4600 to 5200 neutrophiles, and 1600 to 2200 lymphocytes) are averages from several acceptable authors.[1] Because

[1] These modes are the ones in common use. Some recent work by Sabin *et al.* (*Bull. Johns Hopkins Hosp.*, 1925, xxxvii, 14) and by Shaw (*Jour. Pathol. and Bacteriol.*, 1927, xxx, 1) would indicate that the range may be much greater and that during twenty-four hours a variation of considerable degree may take place normally. This, however, does not vitiate the results of this work, because the course of the neutrophiles rather than any single count is the essential feature.

of the variation of totals every chart cannot be made on the same scale, it being obviously impossible to illustrate conveniently on the same scale of drawing a leukocyte number of 300,000 and of 5000.

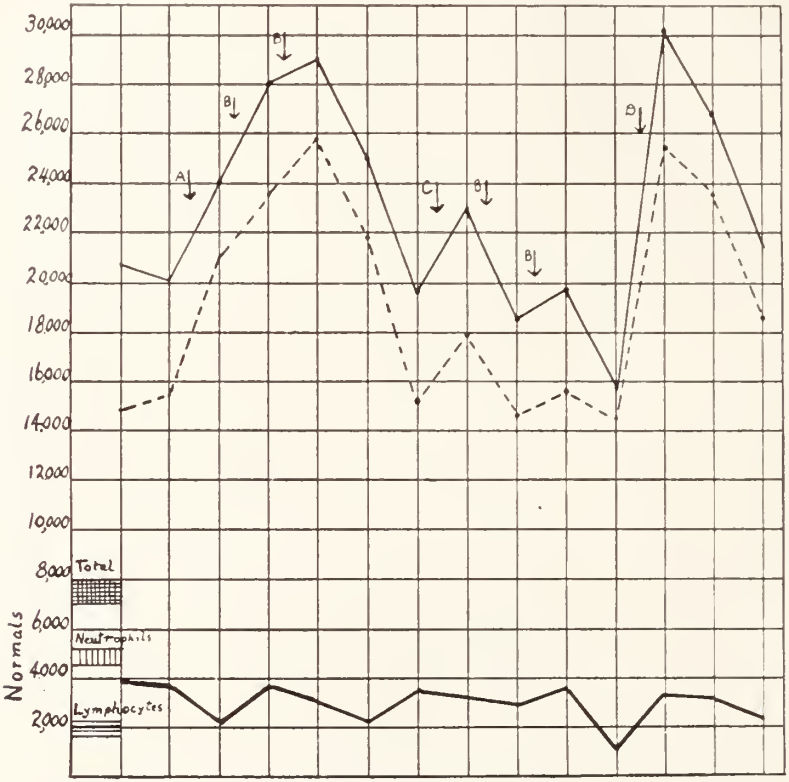

CHART III.—Case III. A low-grade lymphatic leukemia of chronic character, which was never satisfactorily explained on a clinical basis; five weeks' observation; apparently a low-grade infection, of pulmonary origin, in which the lymphocytes were stimulated and the neutrophils depressed; the neutrophils tended to fall (heavy line), and a drop occurred after nearly every roentgen-ray exposure, until they were finally stopped when the number of cells fell to 1200 per cubic millimeter; vaccine and bath therapy did not raise the number of these cells; the number was always below normal; the case certainly belongs near the chronic lymphatic leukemias; *A* indicates roentgen ray begun; *B*, roentgen ray; *C*, vaccine; *D*, roentgen ray stopped.

The basis of all is, however, the same, so that the reader can quickly become familiar with the method. Heavy solid lines indicate neutrophiles, dash lines lymphocytes and light lines are total leukocytes. The black squares indicate death.

The first two graphs, containing the records of three patients, illustrate cases of chronic lymphatic leukemia. The total and lymphocyte counts have been influenced as expected by radiation. The number of neutrophiles is below normal, and the effect of x-ray is to keep it there or to drive it lower. The depressing effect is shown especially in Cases I and II. The number of polymorphonuclears in chronic lymphatic leukemia has long been known as low, but this

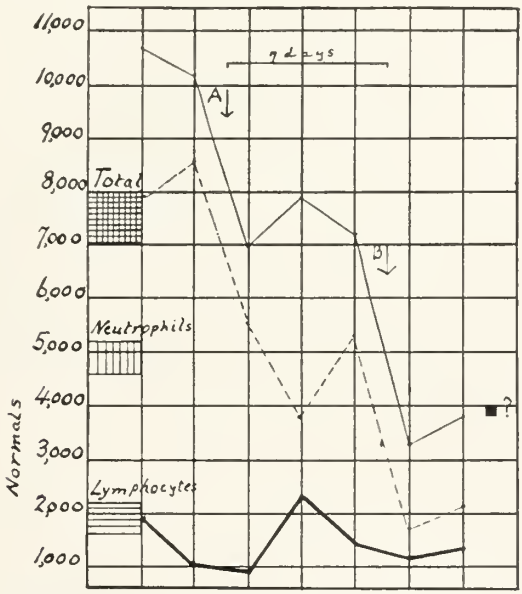

CHART IV.—Case IV. A low-grade chronic leukemia with general adenopathy and slightly enlarged spleen, with severe secondary anemia and low platelet count (chronic sublymphemic leukemia—thrombocytopenic anemia); three and a half weeks' observation; the depression of marrow activity is shown in the number of circulating polymorphonuclears; they were unfavorably affected by the roentgen-ray, which reduced the lymphocyte count to normal proportions; radiation was stopped because the blood did not improve and the patient was in poor condition; *A* indicates roentgen ray started; *B*, roentgen ray stopped.

has not been emphasized; Nägeli in his textbook on the blood does not mention it. In none of these cases did it reach normal at any time.

Case IV is one of low-grade lymphatic leukemia that had a suggestively infectious etiology. The tendency for the neutrophiles to fall all through the course is evident; dips in the count can be seen to follow nearly every exposure to x-ray.

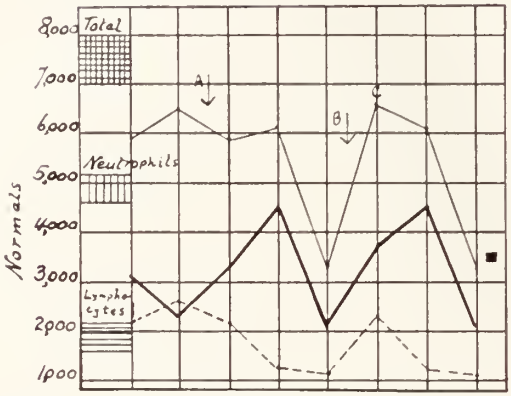

CHART V.—Case V. A case of generalized lymphadenopathy in which the relations of the total leukocytes, neutrophils and lymphocytes remained about normal, but the total numbers were subnormal in most of the course (aleukemic lymphadenosis); six weeks' observation; the neutrophils were variable and did not show the effect of the roentgen ray so much as did the lymphocytes; they were obviously unable to cope with the pneumonia when it developed; autopsy confirmed the diagnosis, showing a picture of lymphatic leukemia with the addition of large masses of leukemic tumors in the retroperitoneal area; the bone-marrow was not studied; if this were to be included with the chronic lymphatic leukemias, it would have to be considered as being studied during an interval when the relations of the important white cells were other than usual for the chronic form of the disease; *A* indicates roentgen ray started; *B*, roentgen ray stopped; *C*, pneumonia.

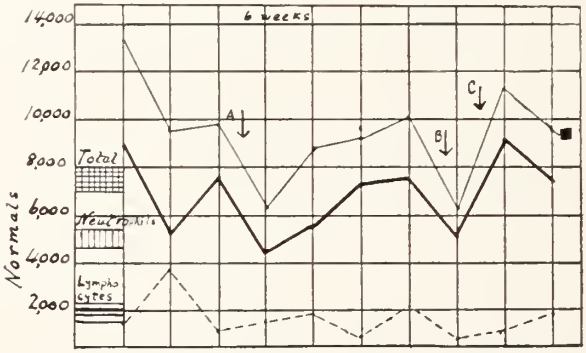

CHART VI.—Case VI. A case of generalized lymphadenopathy without involvement of the spleen and without leukocytosis; the neutrophil count was slightly affected by the radiation treatment; the red cells and hemoglobin remained normal to the end; the last phase consisted of multiple bone tumors that were called myeloma because of the structure of one that was removed at a limited autopsy; the diagnosis was confirmed at autopsy; *A* indicates roentgen ray begun; *B*, roentgen ray stopped; *C*, interval bone tumors.

Cases V and VI are definitely sublymphemic leukemias with subnormal neutrophiles. Case V was not so strongly affected by radiation, but this neutrophile reduction is clearly shown by the first series on the Case VI.

The next three cases, VII, VIII and X, are instances of aleukemic adenoses. In the first one the neutrophiles were constantly low and were affected at one time by *x*-ray. This was just before pneumonia developed, and we may suspect the relationship between this

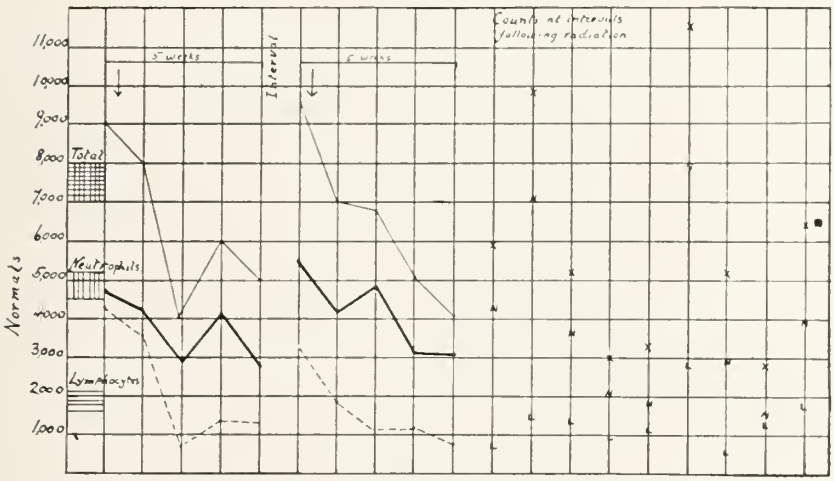

CHART VII.—Case VII. A case of aleukemic lymphadenopathy with a slightly disproportionate lymphocytosis; the lymphocytes were apparently susceptible to the roentgen ray, the neutrophils much less so; this case showed decided lymphadenopathy, with enlargement of the spleen and liver; the whole observation covered two years; the primary growth started in the neck and spread thence to the mediastinum and axillæ, and then the abdomen; blood counts would indicate that at no time did the patient have a leukemia; the terminal picture was that of multiple myeloma; a review of this case seemed to indicate that it was a slowly progressive lymphosarcomatosis; arrows indicate roentgen-ray series.

low count and susceptibility to infection. This case, because of the low neutrophiles and their sensitivity, is one of the borderline cases to which Pinkus has called attention as a possible relative of true leukemia. Cases VIII and X show neutrophiles that try to remain within normal limits and lymphocytes that fall progressively with radiation.

Case IX, one of marked lymphadenopathy with leukopenia and greatly reduced neutrophiles, is one that we feel was not helped by

radiation. Perhaps this was a case of acute aplasia of the marrow and in which lymphocytes were increased, as the neutrophiles failed.

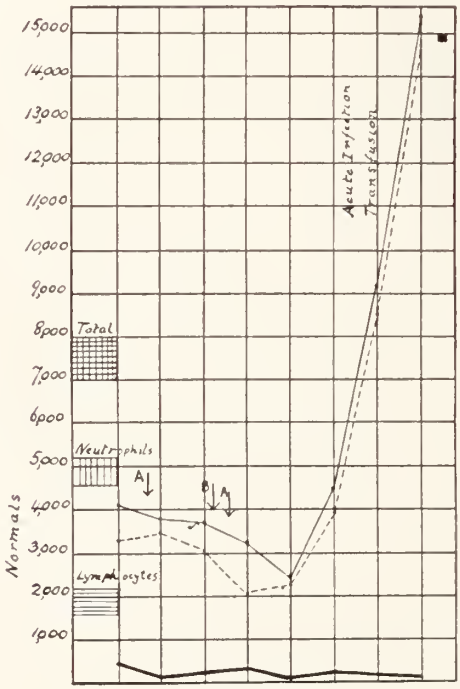

CHART VIII.—Case VIII. A case of general lymphadenopathy with slow failure of bone-marrow activity; nine weeks' observation; it may be noted the neutrophils were hardly ever more than one-tenth of their normal number; the red cells fell in nine weeks from 4,000,000 to 2,500,000 and the hemoglobin from 75 to 50 per cent; during the course of radiation treatment the glands decreased and practically disappeared and the man became much worse; despite transfusions the condition of the blood did not improve; when an acute respiratory-tract infection appeared, the neutrophils did not respond, the rapid rise in circulating leukocytes being entirely due to small mononuclears; autopsy was not performed; *A* indicates roentgen ray; *B*, transfusion.

Cases XI and XII are examples of leukosarcoma. Case XII showed very susceptible neutrophiles, while Case XI tried to make enough but they were apparently knocked out by the radiation. After several exposures the number fell to 1500. It was raised to above normal limits by vaccine, but tended for several days to drop to subnormal figures. Late in the course normal counts were obtained.

Cases XIII to XV inclusive are examples of lymphosarcoma in which the neutrophiles are practically always within expectable variations and show only very transient radiation effects.

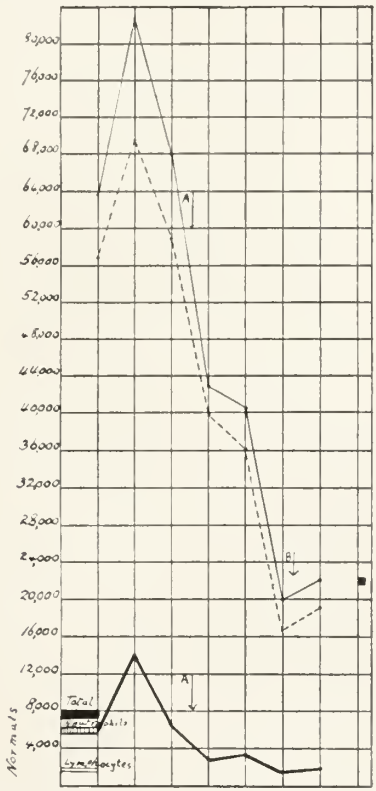

Chart IX.—Case IX. A case of tumor of the mediastinum and abdomen with insignificant enlargement of lymph nodes; five weeks' observation; the mass in the chest was accompanied by considerable fluid; the abdomen was as large as in a full-term pregnancy, only part of which enlargement was fluid; the circulating cells were chiefly adult small lymphocytes; there is evidently a leukemia; this case therefore comes in the group classified as leukosarcoma; it may be noted that the neutrophil count was normal or above until roentgen-ray treatment was started, when it fell to low figures; the patient died after a bronchopneumonia; the neutrophils did not increase during this infection; *A* indicates roentgen ray begun; *B*, roentgen ray stopped.

Average Hodgkin's cases in the early and middle stages are illustrated by Cases XVI and XVII. The neutrophiles, while varying slightly with the total count, remain mostly above normal. They are not driven to subnormal levels.

Cases of Hodgkin's disease in which severity of course and malignancy of pathology are prominent features behave somewhat differently, as shown by Cases XVIII and XIX. The former had distinct

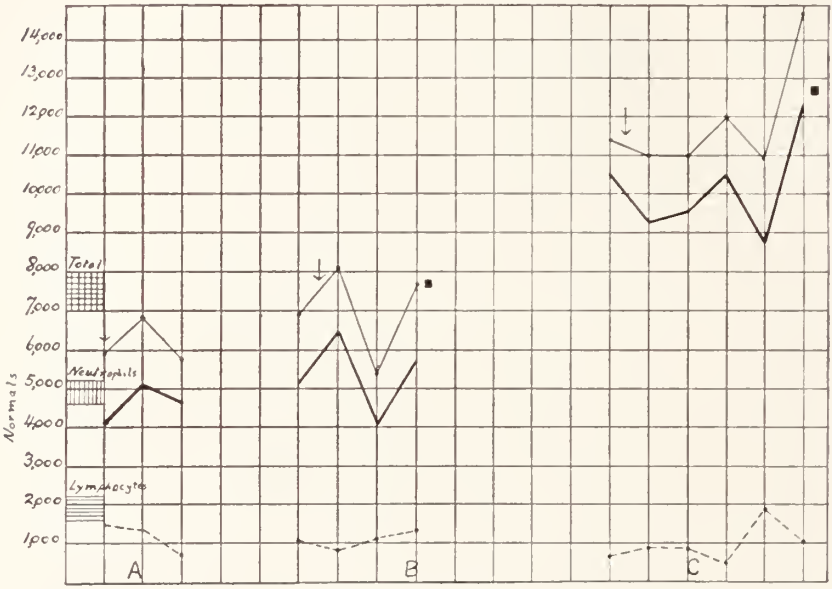

Chart X.—Arrows indicate roentgen-ray series. *A* (Case X), was diagnosed as lymphosarcoma or possibly aleukemic lymphadenosis, of two years' duration, originating in the neck and extending to the chest, and finally to the lower trunk as retroperitoneal and pelvic tumors; three weeks' observation; the blood count during one of the patient's admissions to the hospital is shown; almost identical figures occurred on another admission; the neutrophils were practically unaffected by the roentgen-ray series and the counts were within normal limits. *B* (Case XI), lymphosarcoma of the neck occurring five years after removal of tumor from the neck, five weeks' observation; the final picture was that of lymphosarcomatosis, which was confirmed by autopsy; the counts were made during the final admission and show that the radiation did not have a marked effect on the neutrophils, certainly not keeping them below the normal line. *C* (Case XII), five months' duration, apparently beginning as a tumor of the groin after injury, thence extending to the axilla and to the neck, followed later by signs of growth in the chest; three and one-half weeks' observation; diagnosed as lymphosarcoma because of the picture of excised glands, the blood count and the rapidity of the process; autopsy revealed a large mediastinal growth, isolated nodules in the spleen and kidney and a large mass in the retroperitoneal and iliac region; the blood showed a moderately high leukocyte count with a relatively high neutrophil number; the neutrophils were unaffected by the primary process and not materially reduced if at all by the roentgen ray.

bone lesions, while the history available from Case XIX suggests strongly the same involvement. In both cases the neutrophiles are very susceptible.

Nonlymphoid sarcoma, as illustrated by Case XX, showed no susceptibility of neutrophiles on the first series, while the second series occurred late in cachexia.

Endothelioma, seen in Case XXI, also failed to be distinguished by a special neutrophile number or susceptibility.

SUMMARY. The unwisdom of drawing conclusions from any present knowledge of lymphadenopathies is only too evident, but it would seem that a little further support may be had from the analysis of neutrophiles and their reaction to radiation for the separation of certain groups and for rules of treatment.

From the evidence here collected the leukemic group is composed of true chronic lymphatic leukemia, sublymphemic leukemia and leukosarcoma, in that they have, with lymphocytosis, a low number of neutrophiles which are very susceptible to radiation treatment. Aleukemic lymphadenosis tends to have the percentages and numbers of neutrophiles and lymphocytes approach normal, with relative insusceptibility to radiation. The cases have a tendency to osseous involvement, which is, however, not in our two cases attended by polynucleosis, and therefore suggest myelomata, which are not characterized by a peculiar blood picture. This group of adenoses resembles somewhat in hematology the lymphosarcomata. The tendency to final malignant stages as evidenced by the bony tumors in adenosis increases the resemblance. The neutrophiles in lymphosarcoma do not fall consistently under radiation.

Hodgkin's disease is marked by a polynucleosis which is indifferent to radiation, except in the malignant and febrile cases when these cells are quite variable. Our single cases of reticulum-cell sarcoma and endothelioma had neutrophiles unaffected by radiation and of no definite numbers.

From these figures, then, one can deduce that there is a leukemic group (true chronic leukemia, sublymphemic leukemia and leukosarcoma) and a sarcoma group (aleukemic lymphadenosis and lymphosarcoma) with distinct neutrophile picture and reaction to radiation. These are separate from Hodgkin's disease, which varies with its type and from endothelioma and nonlymphoid sarcoma. These features, if corroborated, will, with a biopsy, somewhat simplify the classification of a given case.

DISCUSSION

Dr. John H. Gibbon: I would like to ask Dr. Fox for a differentiation in the type. Is he prepared to tell us what effect, if any, the reduction in neutrophiles has on the course of disease and on the symptoms? I would particularly like to ask him what effect it would have in an acute lymphatic leukemia. There was one acute case in the series.

Dr. Fox (closing): Do I understand that Dr. Gibbon would like to know the effect upon both types, the acute leukemias and others? The one case to which you referred, probably of acute leukemia, was certainly damaged by the radiation. The reason we say so is because of the fall of hemoglobin percentage, the red cells and the neutrophiles. The mass evidently became much scattered after the treatment was begun. The effect of the fall of neutrophiles during lymphatic leukemia is practically *nil* so far as we can see. We cannot trace any course clinically during the fall. We are afraid, with the absence of neutrophiles, there will be greater susceptibility to infection because of the first 2 cases of which I spoke.

SNAKE POISONING AND ITS TREATMENT*

By AFRANIO DO AMARAL, M.D.

(*Abstract*)

In my communication on "Snakes and Snake Poisoning," I spoke particularly of the three topics, the snake, the venom and the poisoning.

In regard to the first topic, I showed the importance of a thorough knowledge of the different species of snakes, their geographical distribution, relative incidence of species, habitat, and living, feeding and mating habits. I emphasized the economic value of most of the nonpoisonous snakes that, as a rule, feed on mice, rats, moles and other rodents quite destructive to agriculture. By showing a set of lantern slides, I demonstrated not only the probable evolution of the ophidia from the most primitive types to those that have a highly developed poison apparatus, such as, in North America, the copperhead, the moccasin and the rattler, but also the evolutionary changes that have appeared in their maxillary bones, mandible, teeth and fangs. In this respect, the proteroglyph snakes seem to have come directly from the aglyph, while the solenoglyph apparently have evolved from the same group, the aglyph, but indirectly, through the opisthoglyph. I finally showed the mechanism of the bite, and also how far a snake can strike, that is usually about one-third the length of his body.

Concerning the venom, I gave a demonstrate of its extraction and showed how it is prepared for laboratory work and how it affects the different animals. Every venom has a special activity toward the tissues of the various animals used in laboratory experimentation. In this regard, snake poison must be considered the most complex of all antigenic substances known, as from eight to twenty different principles have been found in the venoms so far studied.

* Read, March 2, 1927.

In regard to the poisoning, I showed that the variation of its types are due to the special affinities of the various toxic principles toward the living cells, a few of them affecting the red cells, or the leukocytes, the fibrin, the endothelial cells, the different nerve centers, the peripheral nerves, the neuromotor apparatus, etc. The death-rate varies, too, in accord with the activity of the amount of venom secreted by the different snakes, and also in accord with the condition and age of the persons bitten. It is well known that over 25,000 people die from snake bites in India every year. In this country, the total number of deaths from snake bites seems to be well over 1000 a year, and the death-rate varies in the different zones considered as follows: In the Middle West, along the West Coast and in all the Eastern States it averages 10 to 12 per cent; in the Southeastern States it is between 15 and 25 per cent, which variation seems to be due to the different potentiality and amount of venom secreted of the two main types of snake of that region, namely, the *Agkistrodon piscivorus* (moccasin) and the *Crotalus adamanteus* (diamond-back rattler); finally, in the Southwest, it oscillates between 25 and 35 per cent, the latter figure mostly prevailing in Texas.

The prophylaxis of snake poisoning can be achieved by several means, such as the extermination of snakes either by hunting and killing them directly or by raising ophiophagous animals; the prevention of bites especially by the use of shoes and leggings; finally, the capture of snakes which can be made useful if their venom is extracted and employed in the preparation of antivenins.

In relation to the treatment of snake poisoning, it is known that no mechanical, physical or chemical process so far developed can be recommended in exclusion of the specific treatment, because as soon as the venoms are inoculated into the animal they become so closely attached to the cells by means of chemical combinations that only a substance having a particular affinity for them is able to attack and destroy them. This substance is the antivenin that comes from animals actively immunized against the snake poisoning. In the preparation and administration of antivenins, it is necessary to bear in mind that the principle of specificity must be adhered to. For the active immunization, the venom should be prepared in such a way as to keep all of its principles as this will enable the antivenins to be effective against all of them. Before being given for human application, the antivenins are tested for sterility and antitoxic

activity. This titration can be done on various laboratory animals, among which pigeons are most recommended. Their preventive and curative actions can also be demonstrated on mammals, such as mice, rabbits, etc.

As a result of the apparent increase of the number of bites in this country, which seems to be due to the agricultural development especially in the South and Southwest, the Antivenin Institute of American has been organized as a Division of the Mulford Biological Laboratories in order to cope with the new problem. A few stations have been established in the snake-infected districts with a view to securing as many poisonous snakes as necessary for a continuous supply of venom.

The antivenin is given hypodermically or, in case of severe poisoning, intravenously, in doses varying from 10 to 20 cc. As the different species of Nearctic poisonous snakes, as a rule, do not kill people in less than twelve to twenty-four hours, it is expected that the use of this specific antivenin will result in a sharp decrease of the mortality.

ADDRESS BY HIS EXCELLENCY SIR ESME HOWARD, THE BRITISH AMBASSADOR.*

It is really an exceptional pleasure to come here tonight in order to take part in this celebration to honour the memory of Joseph Lister who is, I venture to think, perhaps the greatest man that England produced in the last century and amongst all Englishmen of his generation the one who has been the greatest benefactor of humanity. I never realized what he had done for the world at large, until, within recent years, I read his biography and understood the state of our hospitals in Great Britain before his time. As I read his biography there came back to me an experience of my very young days. When I was a boy my mother used very often to take me around with her in order to visit the village folk who were ill. I remember around about 1869 or 1870 going to see a man who had broken a leg or an arm. After we came away she said, "It is a great pity that man will not go to the hospital," and I asked, "Why won't he go?" "Well," she said, "they are all afraid if they go to the hospital that they will not come out alive." I confess I wondered very much at the time how this could be. When I was very young I had great confidence in the infallibility of all doctors. That confidence is perhaps not quite so strong as it was, though it is still strong enough, I am glad to say. Anyhow I never realized then what a general feeling, or fear, there was among the poorer patients that if they went to the hospital they would die, until I read the biography of Lister. Quoting from memory, I believe that before his time the percentage of deaths in surgical cases was well over 50 per cent, so that one can well understand why people were afraid to enter the hospitals in those days. That has now, fortunately, all been changed and I often wonder whether there ever was really any man in England who did more to help suffering humanity. Although there were many who were working along the same lines at the same time, I think there is little doubt that Lister will ever be remembered as the first among English

* Read April 6, 1927.

surgeons of his day. I should like also to say this with regard to him. He was great not only for his genius, but also for his character. In many long years of controversy he always remained modest, considerate, like St. Paul's description of charity, kind, never puffed up, not seeking his own. He was, I think, in all respects a great man worthy of our admiration. This Continent is now becoming the center of research, everywhere we read of and see great temples for research going up and great strides being made in advance of science. There is no doubt that humanity has now and will have in the future, much to thank America for. America is in the van for all this movement at the present time. While in the political and religious spheres there are many differences of opinion, in this realm of medical science we have a purer atmosphere where we can all join, whatever our feelings are about other things, in admiration of the great men who have worked to save humanity from unnecessary suffering. Very often when I read certain articles in the press, when I hear certain speeches delivered at political gatherings, I wish to God that some political Lister would rise and would save us from the jealousies, suspicions and irritants that have come into the world and have acted like septic poisoning to the body Politic. Ladies and gentlemen, in honoring the few who have succeeded we must also not forget the many who have given their lives in the cause, who have sought and not found, who have knocked and to whom the doors have not been opened and who have struggled and not reached the goal; they also deserve our thanks. The President has told you I only intended to say a few words tonight, I have made it longer than I intended, but you will thank me for its brevity which is, as you know, the soul of wit. It only remains to thank you for this very kind invitation you have given me this night and on behalf of my fellow countrymen to thank the College of Physicians for honoring the memory of this great Englishman and for inviting me to take part in this ceremony. I thank you.

THE CENTENARY OF LISTER (1827–1927): A TALE OF SEPSIS AND ANTISEPSIS*

BY ASTLEY P. C. ASHHURST, M.D.

Surgery holds three fundamental problems: The control of hemorrhage, the prevention of pain, and the care of wounds. France, by the intrepidity of Paré, has contributed most to the problem of the control of hemorrhage; America, by the introduction of surgical anesthesia, by Morton and by Long, has done more for the prevention of pain than any nation before or since; and Britain, in the person of Joseph Lister, has shown the way along which surgeons must travel to ensure for their wounds an uninterrupted course of healing.

It is for the celebration of the hundredth anniversary of the birth of this great *Surgeon*, that the College of *Physicians* of Philadelphia has invited you to be present tonight, on this which is only the second occasion on which she has given official recognition to surgery and the surgeon. Forty years ago, at her Centennial Banquet, the College gave her first official recognition of surgery as distinct from medicine, when she invited one of her beloved and distinguished sons to respond to the toast, "The Surgeon." As a token of affectionate respect for his memory, a son of that surgeon has been appointed by the President of the College, and by a majority of the Committee on the Lister Centenary Celebration, to address you this evening.

I ask you to journey with me backwards, into the mists and uncertainties of the past of surgery, and to observe and note what were the conditions under which surgeons worked and patients suffered before the dawn of antisepsis. It is a long journey, over rough roads: over roads obstructed by quagmires of putrefaction and morasses of phlegmon; over roads which lead through the stench of hospital gangrene, the miasms of septicemia, the fires of erysipelas, and the chills of pyemia; into the by-paths and impasses of angeioleucitis; and across deserts where the shifting sands of empiricism

* Read April 6, 1927.

Lister

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often covered the main route, and caused surgeons to lose their way. It is a sad journey; and if there were no other prospect at its end, it were best forgotten.

There were great surgeons before Lister, just as there were brave men before Agamemnon. The best of these surgeons possessed a reasonable understanding of the normal processes of healing. They recognized that there was no dressing so suitable to be applied to a fresh wound as the opposite side of the wound from which it had just been severed. They found that in the majority of cleanly cut wounds, union "by adhesion," as it came to be known, could be thus secured if the wound was accurately but not too tightly sutured, after checking all bleeding from its surfaces, so as to prevent the collection of bloodclots between its lips; for the presence of bloodclots or any other foreign substance in the depths of a wound not only prevented union by adhesion, but actually caused suppuration. They had learned that in deep wounds, involving muscle, it was difficult to secure union by adhesion, even though the wound had been closed promptly by sutures; because these sutures had a tendency to unite the superficial parts of the wound, but to allow its interior to gape; and in this gaping interior blood would collect, putrefaction would occur, suppuration would supervene, and the wound would break down and leave a stinking and a painful sore. It is true that some surgeons, very careful and neat in their dressings, were able to apply compresses so skillfully and to use the roller bandage so intelligently, that they could frequently succeed in keeping the deeper parts of these large wounds in contact, and that in many instances they were able to make such wounds unite by adhesion, where their less skillful or less painstaking and attentive contemporaries never could get such wounds to heal unless left wide open from the first.

Though this doctrine of adhesion of wounds appears to have been known in the very earliest days of surgery, the medieval surgeons (if surgeons they could be called) seem to have forgotten it, or to have mistrusted it. In the almost unending wars by which Europe was rent for hundreds of years after the introduction of gunpowder, the great majority of wounds seen by surgeons were frightful in extent, badly lacerated and contused; such, in truth, as could not be made to heal by adhesion even if promptly reunited by sutures. Even sabre wounds, which were frequent in those days of hand-to-hand fighting, were usually very extensive, and always more or

less contused or lacerated; so that all hope of securing union in them seems to have been very quickly abandoned. Thus it was that many surgeons brought back with them into civil life a doctrine of despair; a doctrine to the effect that wounds, especially gunshot wounds, were *poisoned*, and on that account alone could never be expected to heal unless treated by some method to overcome the poison. As a consequence, in the words of the eloquent John Bell, writing one hundred years ago—

"They never allowed the lips of a wound to fall together: they filled it with dressings and acrid balsams, or distended it with tents and leaden tubes. They were ingenious in every invention to prevent its sudden healing . . . Every wound was forced into a painful suppuration, which the surgeons of those days were careful to encourage in proportion as the wound seemed more inflamed. The quantity of matter was again an argument that the wound needed to be thoroughly purged of its foul humors. The pain of a wound and the waste of substance was very great, under their management: the cure was proportionably slow, the scar was broad and unsightly; but they comforted themselves by saying, that this method alone was sure, and consistent with the rules of art. Those few surgeons who ventured upon the cure by direct and immediate adhesion, went fearfully and timidly to work. They were in a continual apprehension of ill accidents; and having failed to comply with the canonical or regular method, having neglected to purge the wound, the patient never suffered the slightest alteration of his health, even at the distance of years, which was not imputed, either from malice or ignorance, to this sudden and improvident cure.

"This" (continues John Bell), "was the real origin of the secret dressings; and nothing can be more singular than the various inventions which surgeons, in those days, fell upon, to persuade their patients to let their wounds be easily healed, and, at the same time, protect themselves from blame."

As Bell points out, the continual protestations by La Motte (1722) and other good surgeons, of their never having, for the sake of gain, used any tents or injections to protract a cure, suggests to our minds some very unpleasant reflections.

"For while these authors enter their protest against the general conduct of other surgeons, and show their anxiety to proclaim their own innocence, we cannot but see that they point at some practices and motives of conduct which we fain would not believe.

"In those days, every flap of skin, instead of being reunited, was cut away; every open wound was dressed as a sore, and every deep one was plugged with a tent, lest it should heal. Tents, syndons, setons, leaden cannulas, and strong injections, were among the chief violences of ancient

surgery. And no man . . . who regarded his own character went without this cruel apparatus of tents and syndons when he was to dress even a slight wound.

"The old surgeons" (pursues Bell) "never put the lips of any wound together: if it was not large and free, their rule was to dilate it, but never with the knife; with a sort of forceps they tore it open; they seldom made counter-openings to let out the matter; they never sought to put an end to the formation of pus and the waste of the fleshy parts, but encouraged the suppurations. They plugged up the orifice, in order to dilate it. They made their tents long, that they might convey acrid medicines to the very bottom of the wound. They forced the suppuration, in order to prepare it for the second stage of their process, namely, incarnation. They prevented the natural and easy flow of the matter, that they might have the satisfaction of seeing it spout out in great profusion when they withdrew their tent. They opposed their own ways to those of nature, and liked nothing which was not done by main force of surgery . . . No wound escaped this violence; and the practice of an ancient surgeon was exactly the same with that by which beggars counterfeit sores, or rather convert accidental wounds into perpetual ulcers. The most simple wounds of the limbs were frequently tortured by the old surgeons into the form of malignant sores running for many months; wounds of the great cavities were always distended with tents to the imminent danger of the patient: if he survived, there remained fistulas which would never heal; the sores ran more or less according to his state of health, or the season of the year; and a man wounded in the chest wore his leaden cannula for life . . . The old surgeons thrust their tents into all wounds, both those which were in danger of healing and those also which could not heal. They thrust them into gunshot wounds, of which, if they had understood the nature, they could have been under no apprehension of their healing suddenly, since the whole course of a gunshot wound is mortified, and the parts through which the ball has passed, being effectually killed, must be thrown in sloughs." (I still quote John Bell.) "They thrust their long tents into the neck, or even into wounds of the cheek, until the neck or head swelled monstrously. Even in compound fractures, they thrust them deep betwixt the ends of the broken bones, as if they had been afraid lest a callus should form. They introduced their tents even into the cavities of the belly and breast. They never trepanned the skull where there was inward suppuration, without thrusting a tent into the substance of the brain itself, though sometimes they were content with laying in a syndon, or slip of linen. 'Often, I am sure,' says La Motte, 'I have seen those who would have been very willing to have taken the help of a mallet to drive their tents into the thorax. It was from shame only that they refrained.' "

There arose, then, as intimated above, a school of secret healers, who attributed their powers of curing wounds, almost or entirely without suppuration, to knowledge gained "from some eastern sage, or to have found out by deep studies in philosophy and alchemy, a sympathetic, or, as they often called it, a philosophical cure of

wounds." By dint of leaving the wound alone, they let it heal naturally, while they amazed the patient and his friends by applying their magic powders and sympathetic solutions to the bloody weapon which had caused the wound; or, if the original weapon could not be found, then to a similar weapon, or even to a stick drawn across the wound, or to a bloody towel which had been in contact with it. But it is to be noted well that these quacks refused to use their magical cures in the case of any wound where they could foresee suppuration; and they absolutely refused to treat gunshot wounds.

Because gunshot wounds were poisoned, it was necessary to kill the poison by pouring over them "oil of elders, scalding hot;" and the story is well known of Paré, who, in 1536, finding after a famous battle that his burning oil was used up before he had poured it into the wounds of all the patients under his care, made bold to use instead "a digestive of yolkes of egges, oyle of roses, and turpentine."

"In the night" (he says) "I could not sleepe in quiet, fearing some default in not cauterizing, that I should find those to whom I had not used the burning oyle, dead impoysoned; which made me rise very early to visit them, where beyond my expectation I found those to whom I had applyed my digestive medicine, to feele little paine, and their wounds without inflammation or tumour, having rested reasonable well in the night: the other to whom was used the sayd burning oyle, I found them feverish, with great paine and tumour about the edges of their wounds. And then" (he adds), "I resolved with myselfe never so cruelly to burne poore men wounded with gunshot."

This same Paré it was, who, about twenty years later, had the courage also to abandon the use of the actual cautery in favor of the ligature for the arrest of hemorrhage in amputations. But the majority of surgeons looked upon him as an obnoxious reformer, and continued to use their burnings, their scalding oil, and their tents, syndons, setons, leaden cannulas and strong injections, lest their patients' wounds should heal too quickly. The names of certain surgeons, however, stand out conspicuous above the herd, because of their more intelligent treatment of wounds.[1]

[1] Fioravanti, in the early seventeenth century, possessed the knowledge of secret lotions; and after the wounds had been washed with these, and their edges apposed, he left them to heal in peace; Caesar Magatus, of Ferrara, in 1616, though he followed the popular prejudice in attributing a poisonous influence to the action of the atmosphere on wound surfaces, yet wrote against tents, and opposed dressing wounds with undue frequency. Belloste, surgeon-major of the Hospital at Briançon, in the Alps, about 1700, published an excellent little book on wounds and wound treatment; and about this same time Dionis and other Paris surgeons began to take a more rational view of the care of wounds.

But it was not until the great reforms in surgery inaugurated by the teachings of John Hunter, in the latter part of the eighteenth century, that surgeons in general returned to simple dressings, and allowed nature to bring about healing in her own way: Healing "by the first intention" of nature being rarely obtained except in small wounds, especially of the face; most clean wounds united by adhesion, while those in which adhesion could not be expected, and those in which this method failed, united by granulation and suppuration, called "the second intention."

After this happy and peaceful period of surgery, when Alanson, Hey, the Bells and John Hunter were the main advocates and exponents, in Great Britain, of union of wounds by adhesion, and when their teachings were being spread in this country by our own Physick, the Father of American Surgery, came the period of the Napoleonic wars, with its disruption of civil surgery, and the invasion of metropolitan hospitals by soldiers with gunshot wounds—large, ragged, and suppurating; and again there came an era of civil surgery when the experiences and teachings of military life prevailed. No surgeon dared to close his wounds or to make any attempt at union by adhesion. The patients' wounds stank and were corrupt; hospital gangrene, erysipelas, pyemia and tetanus again became dominant in every surgeon's mind, and in many a patient's wounds; scarcely any operation could be done with safety in these septic surroundings; and lying-in hospitals became veritable pest-holes, to enter which was almost equivalent to certain death. Seven times as many women died after confinement in hospitals as out of them; ovariotomy was from three to four times as fatal in general hospitals (60 to 80 per cent mortality) as in small institutions or in the patient's home (18 to 24 per cent mortality). Erichsen's figures (1874) show a frightful mortality from sepsis after amputation in all the larger British and continental hospitals: the general mortality after major amputations in civil hospitals varied from 26 to 60 per cent, while in military surgery it rose to 65 and even to 90 per cent (in the Crimean War). Of these deaths, to be sure, many were due to the shock of the original injury, or to that of the operation, which had not taken but merely had failed to save the patient's life. But with all due allowance for such cases, pyemia alone was responsible for the death of from 12 to 30 per cent of patients, counting only those who lived long enough after operation to have a chance to develop this dread disease, and without counting those

additional deaths due to erysipelas, secondary hemorrhage, gangrene of the stump, "exhaustion," or "sudden sinking"—the latter terms probably indicating sepsis of some form as the cause of death.

The cause of this prevalence of hospital diseases was thought to be overcrowding, or ineradicable infection entrenched in hospital buildings. Erichsen recommended that, in every hospital, each ward should be entirely vacated for one month every year, to permit of its cleansing and purification. Sir James Y. Simpson, of Edinburgh, became so ardently convinced of the nefarious atmosphere and hopelessly infected buildings of all large and well-established hospitals, that he advocated tearing them all down and erecting in their stead cottages, widely separated from one another, for use as small hospitals; and he proposed that these cottage hospitals should be of cheap construction, so that it would be perfectly feasible to tear them all down or burn them all up every few years. Simpson found that—

Out of 2089 amputations in hospital practice, 855 died.
Out of 2098 amputations in country practice, 226 died.

Cadge, a well-known surgeon of Norwich, wrote as follows, as late as 1874:

"I have unwillingly and almost tremblingly proceeded to operate in the hospital, but I have had a happy confidence and a perfect assurance that in all private cases I should avoid any of these disastrous consequences . . . I come to the conclusion in my own mind that pyemia, if it does not find its birthplace, does find its natural home and resting place in hospitals; and although a hospital may not be the mother of pyemia, it is its nurse."

But what was it that made these "hospital diseases" develop? All was shrouded in mystery, and most surgeons clung to the theory that the diseases were borne by the atmosphere from one patient to another: epithelial cells, dried pus, and other matters floated off in the air as a "miasm" from these infected wounds, and the impurity of the air caused the wounds of other patients to become similarly diseased because this impure air, when inhaled by the neighboring patients, thoroughly undermined their constitutions.

Erichsen, who published in 1874 an interesting series of lectures on "Hospitalism and the Causes of Death after Operations," founded his argument on the following premises: In his male accident ward, in University College Hospital, London, there were 14

beds; sepsis scarcely ever occurred if no more than 7 patients in this ward had large accidental wounds or had had major amputations, but as surely as the number of such patients exceeded 9, so certainly did hospital gangrene and pyemia appear, even if one or two beds in the ward remained unoccupied. It was clear, he concluded, that the atmosphere could normally absorb, and dispose of, only a certain quantity of miasm arising from wounds; and that, when its point of saturation was reached, the wounds became infected.

"About the contagion of erysipelas" (wrote Erichsen), "there can be no question . . . On January 17, 1851, a case of phlegmonous erysipelas of the leg was accidentally brought into No. 1 ward. As soon as the nature of the disease was discovered, the patient was removed to the erysipelas ward,[1] at the top of the building, having only remained in No. 1 for about two hours. At this time No. 1 ward was perfectly healthy; but a few days afterward a patient lying in the next bed to that into which the erysipelatous patient had been taken, and who had been operated upon for necrosis of the ilium, was seized with erysipelas. On January 22" (continues Erichsen), "I performed five operations on patients who were in this ward. Of these cases 3 were attacked by erysipelas on January 24, namely, a case of necrosis of the tibia, of partial amputation of the foot, and of encysted tumor of the scalp. All these patients died. On January 24 a patient was operated on for strangulated femoral hernia. He was seized with symptoms of low peritonitis, doubtless of erysipelatous character, on January 31, and also died."

It is a frightful picture; and it seemed a state of affairs from which there was no ready escape except to stop doing any operations. In our own city, about this date, Prof. William Gibson was in the habit of announcing every year to his classes at the Philadelphia General Hospital ("Blockley"): "This is the season of the year when erysipelas prevails in the wards; we will do no more operations until the first of March!"[2]

But how was it to be explained that trivial operations might lead to death from erysipelas, while severe compound fractures or dislocations might sometimes escape? Consider the two following case histories, one from Bell, the other from Abernethy, both dated about one hundred years ago:

(John Bell narrates that a slater, having fallen from the top of a house, fortunately lighted upon his right hand, thereby saving his life, but producing

[1] Every hospital had to maintain a special ward for erysipelas.

[2] This story I have from Dr. George W. Norris, on the authority of the late Dr. Andrew H. Halberstadt, of Pottsville, Pa., a graduate of the Medical Department of the University of Pennsylvania in 1853.

a severe compound dislocation at the wrist): "The wrist joint was entirely torn; the radius . . . was terribly shattered; the joint was burst up with such violence that the hand hung quite on one side towards the radius, and turned as it were upon a point; the small end of the ulna, with its styloid process, projected through the wound, and the hand looked as if spitted upon this bone . . . and so violently was the hand distorted that the head of the radius was turned out at the wound, though the wound was chiefly on the opposite edge of the forearm, namely, that next the little finger; along with the radius, one of the carpal bones projected through the wound, quite separated. I cut off the end of the radius" (says Bell),"with the cutting pincers of an amputation case; I dilated the wound with the knife, and I took up one of the arteries with the needle; I snipped away the carpal bone with scissors, for it hung only by a rag of tendon and skin. I turned the hand right upon the remaining part of the fractured radius, laid the palm of the hand and the flat inside of the forearm upon a firm splint, made a stitch in the lacerated skin of the wrist, to hold it together, and finished by laying a large flat sponge upon the open part of the wound, just a little moistened in spirits, so as to make it spongy, soft and pliant. This closed the laceration so smoothly, that almost the whole of the integument adhered; the part which failed to adhere was of no great extent. The matter from it was in small quantity and good. In three days it was plain the hand was safe, and that there would be no gangrene. In about fifteen days the swelling had subsided, the suppuration had become very good, and the parts so close it was likely the callus was beginning to form. This man was about three months under my care . . . and now he finds his hand perfectly useful and strong for work."

Compare with this admirable result of a very severe injury, the following note from Abernethy:

Abernethy operated successfully on a gentleman of stout make, removing from the surface of the pectoral muscle a tumor about 4 inches in length, and 3 in breadth and depth. The tumor "was found to be composed of a steatomatous substance, contained in a thin capsule . . . It was firm and resembled cheese in its yellow color and unctuous appearance, but it was not unctuous to the touch. The wound made in the operation soon healed, and the patient's health was restored to as good or seemingly a better state than before the formation of this disease. He also regained his usual athletic form. But in less than three months of his recovery, two new tumors formed, one above and the other below the cicatrix of the wound. The patient did not particularly attend to them until they had attained a size equal to that of a large walnut. To dissect out both these tumors, and make so free a removal of parts as to render it probable that no new growth would insue, seemed to be a very formidable operation; and, as the nature of the former tumor was known, and it was supposed that these were of the same kind, it was agreed to puncture the upper one, to express the contents, and await the event. This was done by a puncture

of ½ inch in length, made by an abscess lancet. The contents were exactly like those of the original tumor. Vehement erysipelatous or irritative inflammation took place, and sloughing about the diseased part: the inflammation rapidly extended to the opposite side of the thorax, and then down the integuments of the abdomen to the groin. The derangement of the constitution was as violent as the local disease, and in about a week the patient died."

This case, concluded Abernethy, showed the danger of irritating wens. Erysipelas, he held, was a disease dependent upon constitutional causes, especially a deranged condition of the gastrointestinal tract. He never even had an idea that the use of an "abscess lancet" had anything to do with the development of erysipelas. Whether the wound should become infected or not, seemed all a matter of chance.

"A surgeon had to recognize" (writes Godlee), "that there was no security against the simplest and most successful operations being followed by dangerous or even fatal results. When he had closed the wounds, 'he was,' as Volkmann said, 'like a husbandman who, having sowed his field, waits with resignation for what the harvest may bring, and reaps it, fully conscious of his own impotence against the elemental powers, which may pour down on him rain, hurricane and hailstorm.' "

Into this welter of wounds and sepsis came Joseph Lister, as a London medical student, in 1848. He was the fourth child and second son of Joseph Jackson Lister, who, like his father before him, was a successful wine merchant and a member of the Society of Friends (Quakers). Joseph Jackson Lister was in many respects a remarkable man: though largely self-taught, he found time, while actively engaged in business, to indulge, as an avocation, his love of mathematics, gaining "a worldwide reputation for his discoveries in the field of optics, which led to the production of the 'achromatic lens' and the perfection of the modern microscope . . . This work gained for him the Fellowship of the Royal Society in 1832." (Godlee.)

Joseph Lister had been carefully educated under his father's supervision, though his early determination to become a surgeon was not entirely approved. He secured the degree of B.A. from University College, London, before beginning his medical studies,

which he pursued in the medical school and hospital of his Alma Mater. In University College Hospital he became house surgeon under Erichsen, whose studies of hospitalism I have already quoted, and who was at that time one of the leading surgeons in London. Though the use of anesthetics in surgical operations was well established at this time, Lister, in December, 1846, had been present at the first operation performed in London under ether, by Robert Liston, at University College Hospital. The older surgeons continued their habit of rapid operating, displaying great manual dexterity and phenomenal strength, but without the niceties and neatness which were to characterize the technic of the rising generation of surgeons, for whom absence of pain on the part of their patients made hurry unnecessary. The contemporaries and immediate pupils of John Hunter had passed with the last generation—Home and Cline, Astley Cooper and Abernethy, Blizard and Wardrop. Charles Bell and Robert Liston, two of those Scottish surgeons who, as Godlee says, had migrated to London as to a happy hunting ground, had but recently died; but Sir Benjamin Brodie, Sir James Paget and Sir William Fergusson (also a recent arrival from Scotland) were at the height of their fame; while James Syme (who had once established himself in London, but shook its dust from off his feet and retired again to Scotland because he found the professional atmosphere in London distasteful) had already made Edinburgh famous throughout the world: brilliant as operator, inspiring as teacher, beloved as chief, he dominated by his forceful personality the entire field of British surgery; while his colleague, Sir James Y. Simpson, the patron saint of chloroform as an anesthetic, was scarcely less brilliant as a teacher and controversialist, invading all fields of medicine, but practising obstetrics and gynecology. In Paris, Dupuytren, Larrey and Lisfranc had gone, but Civiale, Maisonneuve, Velpeau, Malgaigne and Nélaton, greatest of them all, were at the height of their reputation; while in Germany, though von Graefe and Dieffenbach were no more, Bruns, Chelius and Langenbeck were their worthy successors.

Young Lister, trained by his father in the use of the microscope, showed special interest in physiology, histology, and pathology. Those of his teachers who impressed him most were Wharton Jones, professor of ophthalmic medicine and surgery, but as much interested in physiology as in ophthalmology; and William Sharpey, the professor of physiology, who had been trained as a surgeon under

Dupuytren, Lisfranc and Syme, and had settled in Edinburgh, where he had taught anatomy until 1836—truly a splendid training for his subsequent career in physiology. Sharpey kept in close touch with his Edinburgh friends, spending part of every summer with Syme. When Lister, who from the very first had determined to be a surgeon, had completed his studies at University College and had received in 1852 his degree as Bachelor of Medicine from the University of London, and had been made a Fellow of the Royal College of Surgeons; it was but natural that he should adopt Sharpey's suggestion of spending a month in Edinburgh to observe Syme's practice, before making a tour of the continent and finally establishing himself in practice in London. Little did any of them think that his month of observation was to be lengthened to a residence of seven years in Edinburgh, followed by a period of nine years in Glasgow, and a second residence of eight years in Edinburgh, before he returned to settle in London, in 1877, after an absence of twenty-five years.

When Lister reached Edinburgh, he found much to surprise and to captivate him: he found 200 *surgical* beds in the Infirmary, while at University College Hospital there had been no more than 60; but more charming, surprising, fascinating and incomparable yet, was Syme, then in the zenith of his career as operator and teacher—Syme, with a hot head but a warmer heart; who loved controversy but hated a sham; who was persevering and obstinate, but tender-hearted and compassionate; who was willing to rouse opposition, for he snuffed the battle from afar, and loved a fight—Syme, inspiring enthusiasm, admiration, and love!

Syme learned to love and to trust Lister, and soon the latter was appointed house surgeon to Syme. "It might have been thought," says Godlee, "beneath the dignity of a Fellow of the Royal College of Surgeons of England to take such an appointment; but Lister was amply rewarded." Indeed Syme left the conduct of the wards pretty much in Lister's hands, acting himself rather in the capacity of consulting surgeon; while Lister had, to assist him, twelve dressers, who looked up to him and called him "the Chief." He also busied himself in reporting Syme's lectures for the *Lancet*. In a year's time, Lister became assistant surgeon to the Infirmary; and, after a year longer spent in courtship, he became Syme's son-in-law. But his microscopical and physiological investigations were not neglected: he found it would be his duty to lecture on the pathology of inflam-

mation, and, deciding he did not know enough of the subject himself, he set to work to study it under the microscope in the web of a frog's foot. He was led on from this to a study of the coagulation of the blood.

While these peaceful years slipped by in Edinburgh, all England was sick at heart with the news constantly coming from the Crimean War of the dreadful condition of the wounded soldiers and the pestilential conditions in the military hospitals, where those old, old enemies, whose attenuated forms, as Béclard expressed it, are known as angeioleucitis, erysipelas and phlegmon, were rampant; and where thousands were dying of hospital diseases—hospital gangrene, secondary hemorrhages, and pyemia—where only tens and hundreds had been killed in battle.

But at Edinburgh things went peacefully on, Lister's time being spent in investigation, teaching, operating and practice, until 1860, when, the post of regius professor of surgery in the University of Glasgow becoming vacant, he received the appointment, when thirty-three years of age, after endorsements had been given by many influential friends.

Now Glasgow was a city twice the size of Edinburgh, and the classes in the medical school of its University were very much larger. He took the medical students by storm; they became enthusiastic; they made him honorary president of their medical society. Indeed, as Mumford pointed out, Lister won through life golden opinions from those with whom he came in contact.

But the Glasgow Infirmary, to which he became surgeon, was notorious for its unhealthiness: it contained about 400 beds in all, and one operating theater in which about 300 operations were performed annually. The wards were overcrowded, and had often to be closed on account of the frightful mortality. Lister, after his return to Edinburgh, wrote as follows of his early experiences at Glasgow:

"At this period I was engaged in a perpetual contest with the managing body, who, anxious to provide hospital accommodation for the increasing population of Glasgow, for which the Infirmary was by no means adequate, were disposed to introduce additional beds beyond those contemplated in the original construction. It is, I believe, fairly attributable to the firmness of my resistance in this matter that, though my patients suffered from the ills alluded to in a way that was sickening and often heartrending, so as to make me sometimes feel it a questionable privilege to be connected

with the institution, yet none of my wards ever assumed the frightful condition which sometimes showed itself in other parts of the building, making it necessary to shut them up entirely for a time."

Syme's wards at Edinburgh were comparatively healthy; and he had the proud record of twenty amputations of the thigh for disease, without a death; and of only one death (and that from pyemia) among 40 cases of ligation of the femoral artery for popliteal aneurysm. But he had once said to Lister that he thought perhaps it would be well if immediate amputation were done in every case of compound fracture, since on the whole the mortality would be less than if an effort were made to save some of the limbs. And in Lister's own wards at Glasgow, compound fractures did so badly, that, as noted above, he was often sick at heart. He became convinced that it was not the air alone that was poisonous to these wounds, or rather, as others also before him had suggested, that it was not at any rate the oxygen in the air that caused the wounds to become infected. He remembered the teaching of John Hunter, that if a compound fracture could be made to heal under a scab it would do as well as a simple fracture: and yet the only obvious difference between the two was that a compound fracture was open to the air, while a simple fracture was not.

Semmelweis, the Hungarian obstetrician, practising in Vienna, before the middle of the last century, had made an important observation, and had acted upon it with heaven-inspired empiricism: in one division of the lying-in hospital, where he was assistant, male students attended; in the other division, only midwives. In the division visited by the male students the mortality from "puerperal fever" was about three times higher than in the other division. Semmelweis, as ardent in pathology as in practice, observed in the postmortem examination of the body of one of his colleagues, who had died of septicemia following a wound acquired at autopsy, that the lesions were precisely those which he had been in the habit of finding in the cases of puerperal fever. The truth flashed across him that the medical students, coming from postmortem examinations, were infecting the patients in the wards by their examinations. He then insisted that the students purify their hands in solutions of chloride of lime. The result was an immediate drop in the mortality, much below that of the other division. But Semmelweis was as the voice of one crying in the wilderness: he was ridiculed, persecuted, even driven out of Vienna. Returning to Buda-Pesth in 1850, with his sensitive nature crushed by this contumely, he could not be induced to publish his views in book form until 1861, after repeated solicitation by a few firm friends and believers—notably Rokitanski, Skoda, and Hebra. But he was attacked afresh by his opponents: he was called

"der Pesther Narr," and even Virchow sneered at him as "der Kerl der speculiert." Finally his mind gave way, and in 1865 he was brought back to Vienna to be put in restraint. By the irony of fate he died a few days later "of blood poisoning following a wound in his finger inflicted in the course of his professional work." (Godlee.)

Lister, who was not a great reader, and who was unfamiliar with the continental literature, had never heard of Semmelweis at this period when he was worrying about his compound fractures in the Glasgow Infirmary. Lister's colleague, however, Dr. Thomas Anderson, the professor of chemistry, called his attention in 1865 to the work of Pasteur, who had shown that the air was full of living germs, and that these are carried on particles of dust floating in the atmosphere; that the activity of these particles of dust can be destroyed by heat, that they can be filtered off by cotton wool, or intercepted in the finely drawn-out or tortuous necks of flasks, through which free ingress and egress of air take place owing to the diurnal variations in temperature (Godlee). Pasteur further had shown that fermentation was caused by certain minute germs, that putrefaction was a species of fermentation, and that both might be prevented from occurring in fermentible or putrescible substances if the latter were kept free from these impurities of the atmosphere. At once it occurred to Lister that decomposition in wounds could be prevented, without excluding the air, "by applying as a dressing some material capable of destroying the life of the floating particles." Looking around for such an agent, his attention was drawn to the means of disinfecting by means of carbolic acid (recently introduced into commerce) the sewage of the town of Carlisle, which had proved singularly successful. So Lister applied lint soaked in carbolic acid to some cases of compound fracture: in the cases with only a small external wound healing was usually uneventful; but in those with larger wounds, where more carbolic acid was used, though few of the phenomena of inflammation developed, yet a discharge of pus occurred. This pus, he soon realized, was a new kind of pus, being the effect of the tissue dissolution caused by the carbolic acid; it was not even the "laudable pus" which surgeons had been so pleased to see as an evidence that the patient's tissues were reacting well to the injury, knowing as they did that when the stage of suppuration had been reached the patient had passed the dangers of early death from fulminating sepsis. This new kind of pus was an aseptic pus; but it was undesirable, as interfering with healing,

when collecting beneath the carbolic scab; moreover, the carbolic acid burned the surrounding skin surfaces. So the first improvement was to reduce the strength by diluting the carbolic acid with oil; the next step was to use a putty impregnated with carbolic acid as an external dressing. At first he continued these antiseptic applications only until granulations commenced, returning then to the water dressings which were in common use by other surgeons. But having learned that granulating wounds, even when secured by the antiseptic early treatment, were not entirely immune to the development of hospital gangrene, he soon continued his antiseptic dressings until the wounds were almost or entirely healed. Within a year he was using a lotion of carbolic acid (1 to 40 of water) to wash the skin preparatory to operations, soaking his instruments in a 1 to 20 carbolic solution, and cutting his ligatures short, leaving them to be dissolved by the tissues and absorbed or discharged as the case might be; and he could record that during the last nine months not a single case of pyemia, hospital gangrene, or erysipelas had occurred in his wards.

But most of Lister's contemporaries did not recognize the fact that he was contending for a principle in surgery; they looked upon his innovations as nothing more than the introduction of some new form of wound dressing—and the number and variety of wound dressings already in use was legion. The "open treatment," which consisted in applying no dressings at all, leaving the wound wide open so that its discharges might have ready exit, had its partisans in Britain, as well as in America, in Germany and elsewhere; while a number of French surgeons believed in the total exclusion of the air, Jules Guérin and Maisonneuve adopting aspiration of the air from beneath the dressings after they had been applied, and Alphonse Guérin employing a huge thickness of cotton wadding totally impermeable to the air. The "simple dressing" employed by my father, than whom no one secured better wound healing in these preantiseptic days, consisted in lint saturated and kept moist with pure laudanum, the use of which he had learned from "that excellent surgeon, Dr. Joseph Pancoast;" after forty-eight hours diluted alcohol was substituted and continued until the wound was nearly healed.

Their objections to Lister's methods were, above all, that they were always being changed: first it was carbolic acid; then carbolized oil; then the carbolic putty; then watery solutions; then a carbolized

cerate plaster; then a "lac plaster;" then oil of eucalyptus: Was there to be no end of innovations? Moreover, he never published statistics of his cases; it is true he published, at rather tedious length, individual case histories, with wordy comments; but let him come out and report a series of amputations at least as good as Syme's series of twenty thigh amputations for disease without a death, a series secured without any antiseptic trouble or carbolic acid. What is more, they would contend, this man Lister really does no better then we do ourselves: we have all of us cured cases of compound fracture; we have all of us had periods of nine months or more without a death in our wards from pyemia. Why should we change our methods of dressing wounds merely because he tells us to? Such, I imagine, were the thoughts that ran through the minds of many surgeons when their attention was called to Lister and to his work; and many of them spoke their thoughts aloud, and published their opposing views in the journals.

Meanwhile, in 1870, Lister returned to Edinburgh, to succeed Syme in the chair of clinical surgery. There it was that he added fuel to the fire of discontent by introducing the use of the carbolic spray both during operations and whenever a wound was to be dressed. His theory was that the spray—so fine that it barely moistened a piece of slate held in its path—would kill the germs floating on the dust in the air before they had a chance to alight on the wound. The facts that the patients, and often the assistants and sometimes the surgeon, developed mild grades of carbolic-acid poisoning, and passed smoky urine; that their hands became numb and chapped from working in the carbolized atmosphere; and that occasionally the spray was misdirected by the inadvertence of the assistant who manipulated it, and that it squirted carbolic acid into the eyes of the patient even when she was Queen Victoria; these no doubt were objections, but were entirely outweighed in Lister's mind by the supposed advantages. And the strange thing is that there were so many surgeons the world over who came to agree with him, and blindly adopting the spray as a necessary part of the "antiseptic system," looked upon their colleagues who would not use it as hopeless old fogies. In after years Lister himself wrote as follows: "As regards the spray, I feel ashamed that I should have ever recommended it for the purpose of destroying the microbes of the air. If we watch the formation of the spray and observe how its narrow initial cone expands as it advances, with

fresh portions of air continually drawn into its vortex, we see that many of the microbes in it, having only just come under its influence, cannot possibly have been deprived of their vitality." Yet he continued to use the spray in all operations and dressings from its introduction in 1870 until the year 1887.

But some surgeons were curious enough to journey to Scotland in order to see for themselves what he was doing. Among them was Lucas-Championnière, a young Paris surgeon, who came, who saw, and who was conquered, in 1868. He returned to France, and with clarion voice and trenchant pen gave to the world the first systematic account of antiseptic surgery, *Chirurgie Antiseptique*, Paris, 1876.

The German surgeons, especially Thiersch at Leipzig, Volkmann at Halle, and Nussbaum at Münich, eagerly took up the antiseptic system, as did the best of the Scandinavian surgeons; and everywhere it was the same story, of wards reeking with sepsis soon converted into models of healthfulness: where no one had dared touch a scalpel to a patient, perhaps for months at a time, almost at once pyemia, erysipelas, and hospital gangrene became things of the past; secondary hemorrhages ceased to occur, and patients no longer died of exhaustion or sudden sinkings! Ernst von Bergmann, succeeding Langenbeck at Berlin, in 1882, had the acuteness of vision to perceive that antiseptic surgery was only a transition stage; and that what Lister really had in mind, as an ideal, was aseptic technique. With characteristic German thoroughness he set himself to develop means for securing this end; and with palatial operating amphitheaters, limitless supplies of sterile gowns and dressings, and just enough antiseptics to sterilize the skin of patient and of surgeons, he showed that in most operations healing of wounds can be secured without infection, and without the contact of antiseptics with the open wound. About the same period Terrier was creating a revolution in hospitalization in France, and was teaching by precept and example the highest type of aseptic surgery.

In 1875–1876 Lister made a tour of inspection on the continent, to see for himself the workings of antiseptic surgery in the hands of others; and in 1876 he came to this country, in order to attend the meeting in Philadelphia of the International Medical Congress, of whose surgical section he was elected president. Up to this time antiseptic surgery had made little impression on American surgeons. Nor can it be surprizing that to many it did not appeal: they were not entirely convinced of the germ theory of disease, and except in

the military hospitals during the War of the Rebellion they had not had much experience with these dread hospital diseases. There were theoretical objections of another kind, also: even granting that microbes were the cause of suppuration, it had been proved that microbes could and did exist in wounds, treated by most rigorous antiseptic methods, without producing suppuration and without interfering with the clean healing of the wound. It must be remembered that the science of bacteriology was at this time not only in its infancy, it might be considered as being only in a fetal or embryonal stage of development. The existence of nonpathogenic bacteria was scarcely recognized, nor had Metchnikoff yet demonstrated (1883–1893) the existence of "phagocytosis." Moreover, it was asserted that microbes could be carried by the blood; and if this were so, what was the use of applying antiseptics externally to protect the wound from microbes in the air, while nothing could be done about those brought to the wound by the blood? There were also those who based their skepticism on more practical grounds: they pointed to long series of major operations which they had done with their own hands, with a mortality which was lower by far than most of their contemporaries, and which was as low as or even lower than that secured by the devotees of antiseptic surgery in their first essays.

Thus my father, who was slower almost than anyone else in adopting antiseptic principles, pointed, in 1881, to the fact that for the past ten years he had not had a death from pyemia following a major amputation; and that in his first 100 major amputations there had been only 4 deaths from pyemia, none from erysipelas and none from hospital gangrene. When he compared his mortality of 4 per cent due to pyemia with Erichsen's tables, he found in the latter nearly 14 per cent of patients dying from pyemia after major amputations, or nearly 4 deaths from pyemia to every 1 due to the shock of the injury or the operation.

He could point also to a series of 46 cases of excision of the knee, done without antiseptic precautions, with only 4 deaths (2 from pneumonia, 1 from bedsores, and 1 from secondary hemorrhage), a death-rate of 8.6 per cent, compared to a death-rate of from 24 to 30 per cent from the same operation in collective statistics, which always show too favorable results. He noted that Ollier, the great bone-surgeon of France, having secured only 1 operative recovery among 7 cases of excision of the knee (a mortality of 85 per cent), had abandoned the operation until the advent of antiseptic surgery

encouraged him to adopt it once more: even then Ollier found his mortality (up to 1883) was 33 per cent; while with continued and probably more rigorous antisepsis Ollier reported that among 41 patients whose knees he excised between 1885 and 1891 his mortality was 7.5 per cent—or only 1 per cent less than my father's death-rate without antiseptic methods. It is true that my father was extremely cleanly and neat in his work, and that he used laudanum, alcohol, potassium permanganate and other antiseptics long before finally adopting the "antiseptic system" as it had come to be modified by all sensible surgeons (Lister included) in the late eighties. Six years later, my father wrote:

"I feel compelled to say that while I cannot subscribe to the extravagant laudations which this plan receives at the hands of its more enthusiastic advocates, I believe that, when used with judgment, and, if I may be pardoned the expression, when *diluted with common sense*, it is capable of affording very valuable aid to the surgeon. I have not, indeed, found any marked diminution in the mortality after operations by its employment, but I find that the average period of convalescence is shortened; that the violence of the traumatic fever and the frequency of secondary fever are both lessened; that upon the whole the comfort of the patient is promoted; and that the labor and anxiety of the surgeon are very materially diminished. For all of which I am duly thankful."[1]

These objections, both theoretical and practical, were hard to answer. Lister was like the man who had escaped out of Plato's cave, and had made his way up to the light: when he had seen a vision of his ideal, he came stumbling back again into the cave to tell his former fellow-prisoners of the wonderful things to be seen in the land of promise. Is it any wonder, as he came back with his eyes half blinded by the light of day, that he looked to them clumsy and awkward and ridiculous; and that his first attempts to show others what could be done in the land of vision sometimes turned out to be failures? Lister could himself operate on seven recent cases of fracture of the patella without a death; he could resect a femur for malunion: such operations no one could have done successfully without antiseptic surgery. But, even while at

[1] That the antiseptic treatment of wounds was not always successful in these early days is evidenced by a story well known in the University Hospital at that time. A colleague asked my father's ward surgeon, "How many infected wounds have you on your side of the ward this morning?" And received the unexpected reply, "Just thirteen less than there are on your side the ward!"

Halsted, at the Johns Hopkins Hospital, had, about this period, suppuration in from 7 to 10 per cent of his hernia operations, in spite of careful aseptic precautions.

Edinburgh, Lister found that such cases proved successful only when he did all the dressings with his own hands.

There came, finally, in 1877, a call for him to go to London, to succeed Sir William Fergusson as professor of surgery in King's College. The temptation was not as great as it would have been some years earlier. He was now fifty years of age; he was comfortably and contentedly established in Edinburgh, with a large circle of friends and acquaintances. He was at the top of the world in surgery. Students thronged to his classes; visitors from all parts of the world came to see his clinic and observe his practice. Why should he give all this up? But, up yonder was London, the world metropolis, still unbelieving in antiseptic surgery; surely he must have much to answer for, to his conscience, if he let slip this opportunity of carrying the war into the enemy's country. For Lister was deeply religious, and with a simplicity of character that made him particularly rigorous in regard to what he considered his duty: here, in Edinburgh, was comfort and content; there, in London, was the unregenerate race of surgeons. Was it not his duty to go? Can we not hear the still, small voice of conscience asking him, time and again, "Watchman, what of the night? Watchman, what of the night?" Was it not his duty to carry antiseptic surgery into that night of indifference and too willing ignorance? Must not his conscience have asked:

> "Watchman, shall its beams alone
> Gild the land that gave it birth?"

and can we not hear the answer:

> "Ah, no! Ages are its own—
> Lo, it bursts o'er all the earth!"

And so Lister went up to London. Back into that welter of wounds and sepsis, determined to show the London surgeons what a thing this antiseptic surgery was. Whether they should hear, or whether they should forbear, he was determined. "For," he must have said, "Woe unto me, if I preach not the Gospel."

And yet, his arrival in London created scarcely a ripple in the stagnant pool of professional self-esteem in which the London surgeons were steeped. He found King's College Hospital small, compared to the Royal Infirmary at Edinburgh; the students were not the same enthusiastic young men who had crowded his courses

in Scotland; they rather looked askance at his teaching and practice, which they knew were not in favor at the Royal College of Surgeons. But amidst all these discouragements he kept resolutely at work, and his contemporaries finally grew used to him. With Watson Cheyne as his able lieutenant, he slowly, but surely and progressively, won over the rising generation. His own contemporaries were courteous and friendly, but their surgical habits were fixed; and it remained for the next generation to carry the leaven of antisepsis with them into other metropolitan hospitals and down into the provinces.

From about the year 1890 onward, aseptic, as distinguished from antiseptic surgery came to the fore; and though it is said Lister looked with regret on this innovation, with its parade of sterile gowns and linen, its autoclaves, its steam sterilizers and its marble operating rooms, yet it has proved its right to an honored place in modern surgical technique.

When Lister was about to operate, he first placed the marine sponges and the instruments in carbolic solutions, took off his coat, rolled up his shirt-sleeves, pinned an unsterilized towel over his waistcoat, to keep it clean, and dipped his fingers in carbolic lotion. Then, while the patient was being put under the influence of chloroform, he swabbed the part to be operated on with carbolic lotion, turned on the spray, and was ready to begin. After abandoning the spray, he always surrounded the operative area with towels wrung out of carbolic solution. "During the operation, which was seldom short, for Lister was a slow operator, the hands of everyone concerned were frequently dipped in 1 to 40 lotion. The sponges, when soiled, were washed in cold and hot water and replaced in the 1 to 40 lotion. Thus, although they were forcibly wrung out before being used again, a small amount of carbolic acid was constantly conveyed to the wound; and, as a safeguard against any gross contamination owing to the carelessness of assistants or nurses, he used to wash the wound with 1 to 40 lotion before sewing it up" (Godlee). For ligatures he used specially prepared catgut, brought dry to the operation, and simply soaked for about half an hour in 1 to 20 lotion. The sutures (silver wire, silkworm-gut, or sometimes fine silk or catgut) were prepared only by means of carbolic acid. The dressing, in later years, was a thick pad of gauze prepared with the double cyanide of mercury and zinc: the incision itself was protected from this irritating chemical by the "green protective" (oiled silk covered with copal varnish), which was dipped in car-

bolic lotion just before its application; the deeper layers of cyanide gauze were freshly wrung out of 1 to 40 lotion. "In order to prevent the discharge from soaking straight through opposite the wound and in order to force it to travel to the edge of the dressing before it could be contaminated by germs, a layer of thin mackintosh cloth was placed" between the outer layers of the gauze dressing. All his wounds were drained, as the use of strong antiseptic solutions provoked considerable discharge.

As late as 1905 I saw precisely this method of operating (with the exception of the spray) still in use by two of Lister's most earnest admirers: Annandale in Edinburgh and Lucas-Championnière in Paris.

What a contrast to the modern aseptic methods of operating! Here the instruments, dressings, etc.,[1] are sterilized, the former by boiling, the latter by dry heat, and the instruments are laid on a table covered with sterile sheets. The surgeon and his assistants, in white operating suits and with cap and mask, scrub their hands in soap and water for ten minutes by the clock; they then rinse them well in alcohol and don sterile gowns and gloves. The patient's skin, similarly prepared twenty-four hours in advance and kept covered with sterile dressings, is painted early on the morning of operation, and again on the operating table, with iodin or picric acid in alcohol; and the part to be operated on is suitably draped in sterile sheets and towels, only the immediate operative area being left exposed. As soon as the skin incision is made, the skin edges are clipped to sterile towels, and all skin is thus excluded from the wound. With a fresh sterile knife the surgeon proceeds, using instrumental technique as much as he can. No antiseptics whatever are used during the course of the operation: everything coming into contact with the wound is sterile, and it depends on the unceasing and seemingly pedantic precautions of the surgeon to *keep* the wound aseptic. If one misstep is made, the aseptic has to be abandoned for the antiseptic method; and it cannot be denied that adherence to a strictly aseptic technic is much more difficult, and that many surgeons seem incapable of practising it thoroughly. The antiseptic method is applicable to all primarily septic conditions (many compound fractures, necrosis, abscesses, malignant tumors, amputations, etc.); and is valuable in a somewhat modified form in all operations where the tissues are much bruised or long exposed

[1] Proper bacteriologic examinations are requisite for control of the sterilization of dressings, linen, etc.

to the atmosphere during the course of the operation (some excisions, ununited fractures, tedious dissections, etc.). When, however, the operation is of short duration (under half an hour), or when the tissues, even during a longer operation, are not bruised or otherwise unduly injured, and especially in visceral surgery, the aseptic method is superior.

When either method is properly employed, the wound heals without noticeable inflammatory reaction, no stitch abscesses form, no discharging sinuses remain, no ligatures are slowly eliminated from its depths, no granulations persist at one end of the incision, the comfort of the patient is enhanced, and the after-treatment much simplified.

Time would fail to tell of the many departments of operative surgery which have been developed only because Lister paved the way by his teaching of the proper care of wounds. Other surgeons since his time have made perhaps temporary name and fame for themselves by introducing one or other new antiseptic. During the great German War, Mencière of Rheims introduced, much as had Lister, various lotions and an antiseptic putty; Rutherford Morrison originated a whole system of antiseptic treatment with his bismuth, iodoform, paraffine paste (Bipp); Dakin's solution and Eusol (Edinburgh University solution) have each their advocates still. Lemaitre showed that early *débridement, excision*, and *épluchage*, followed by the free application of iodine and provision for dependent drainage, permitted most war wounds to be sutured, and that, if the patients were not transported, about 85 per cent of these wounds would heal promptly; and it was further found, if wounds so treated were left unsutured at the front in the case of patients who had to be transported, that when all wounds not involving bone were sutured promptly on arrival at the base, about 85 per cent would heal without further difficulty. But all these methods are merely modifications or applications of the antiseptic principle in surgery which we owe to Joseph Lister.

Out of the morass of phlegmon, out of the quagmire of putrefaction, back from the devious paths of angeioleucitis, safely through the fires of erysipelas, up from the shifting sands of therapeutic empiricism, on to the broad firm plateau of antiseptic surgery, this great man has led the nations of the world. Friends, Fellows of the College, let us honor tonight and forever the memory of Joseph Lister, who made straight in the desert of sepsis a highway for those who come after.

THE RELATION OF INDOOR ATMOSPHERES TO HUMAN HEALTH AND COMFORT*

By C.-E. A. WINSLOW, Dr.P.H.
PROFESSOR OF PUBLIC HEALTH, YALE SCHOOL OF MEDICINE; CHAIRMAN, NEW YORK COMMISSION ON VENTILATION

When the wise king of the Hebrews sought to express the fundamental unity of the problems which confront each human life, he began with the atmosphere. "When I was born," he says, "I drew in the common air, and fell upon the earth, which is of like nature, and the first voice which I uttered was crying, as all others do." "The common air" is indeed the basic condition of our existence, controlling it in more ways than are yet fully realized.

The atmosphere of the earth is a layer of gases, of continuously decreasing density as one passes outward from the surface of the earth itself, and of varying composition when one reaches a distance where the decrease of gravitational force is so great as to show a marked differential effect upon its various constituents. Taking the atmosphere as a whole, it has been estimated that it is made up of nearly 4,000,000 billion tons of nitrogen (73 per cent by weight), over 1,000,000 billion tons of oxygen (23 per cent by weight), nearly 160,000 billion tons of argon and other inert gases (3 per cent by weight), and over 50,000 billion tons of water vapor (1 per cent by weight). Since water vapor is so highly variable a constituent, it is usually omitted from statements of the normal composition of the atmosphere; and this normal composition, at or near the surface of the earth, may be stated in the usual form as nitrogen, 78.1 per cent by volume, oxygen 20.9 per cent by volume, and argon 0.9 per cent by volume. Carbon dioxide is normally present to the amount of 0.03 per cent and the inert gases other than argon (neon, helium, krypton, xenon) in still smaller proportions. In addition to these substances, which are relatively constant, there is present a variable amount of water vapor. The maximum amount of moisture which the air can retain in gaseous form varies widely with the tempera-

* Read May 4, 1927.

ture. Thus air at 0° C. will hold only 4.8 gm. of water vapor per cubic meter; at 20° C. 17.0 gm. and at 30° C. 29.9 gm. In view of this fact, we commonly express the amount of moisture present, not in terms of absolute volume, but in terms of relative humidity—the ratio of the water vapor actually present to the amount which the air could hold at its actual temperature if saturated.

There is actually more water vapor in air at 30° C. when it is only 20 per cent saturated (about 6 gm.) than in air at 0° C. when it is 100 per cent saturated (4.8 gm.); but the warmer air of lower relative humidity will be much more avid in absorbing moisture from the body or other moist surfaces and will thus be much more drying in its physiological effects. Desert air and the air of heated rooms in cold climates may fall as low as 10 per cent of their potential saturation value.

In addition to its various gaseous constituents the atmosphere usually contains a greater or less proportion of minute suspended particles in the form of dust; and finally, in even the briefest review of the composition of the atmosphere, we must mention living micro-organisms, present only in very small numbers in pure outdoor air, as Pasteur demonstrated when he opened his flasks of putrescible liquid upon the Mer de Glace, but often rising to several thousands per cubic meter indoors and, when considerable contaminated dust is present, even to much higher figures. In the case of certain industrial processes the atmospheric dust present may contain toxic substances (such as lead) or siliceous particles which play a deadly rôle in the production of industrial tuberculosis. The dust of the air of an ordinary occupied room is, however, of an amount and kind which has no sanitary significance. Nor need we concern ourselves with the bacterial content of ordinary room air. Mouth spray, as Trillat has so conclusively shown, is indeed a fertile source for the spread of disease germs; but this is a problem of contact rather than a problem of ventilation. The droplets of mouth spray fall to the ground very quickly as shown by Robinson and the writer many years ago; and the general atmosphere is in no sense a vehicle for the long-distance transmission of disease.

As a result of the vital processes of the human body or of the combustion of ordinary illuminants (both processes being essentially similar oxidations of organic matter), five changes are effected in the air of an occupied room. The oxygen is decreased, the carbon dioxide is increased, the temperature and moisture are increased,

and there are added to the atmosphere certain incompletely oxidized substances, often of an odoriferous nature. In the case of the human body, the latter materials include the products of organic decomposition discharged upon the skin by the sweat glands and the sebaceous glands (valeric, butyric and caproic acids, methylamine, trimethylamine, etc.); the results of putrefaction of organic substances accumulated through lack of cleanliness of the surfaces of the body and the clothing or arising from the mouth and teeth, and products originating in the digestive canal (intestinal gases, ammonia, hydrogen sulphide, indol, skatol, fatty acids, etc.). It is such products which give to the air of an unventilated room its characteristic "body odor."

Taking up, one by one, these five changes in the atmosphere, produced by human occupancy, the decrease in oxygen which takes place in an ordinary unventilated room was shown by Lavoisier in 1777 to be without practical significance; and in 1862 Pettenkofer showed that the increase in carbon dioxide was equally unimportant. In the deep parts of mines where carbon dioxide has largely replaced oxygen, or at high altitudes where the partial pressure of oxygen has been reduced in similar degree, men actually suffer from oxygen starvation. In the worst ventilated rooms on the earth's surface, however, the natural ventilation through cracks and diffusion through the actual substance of walls and ceiling is so great that oxygen never falls below 20 per cent and carbon dioxide rarely rises above 0.5 per cent. Such changes as this are far below the threshold of physiological significance.

Pettenkofer himself believed that the effect of bad air was due to hypothetical organic poisons associated with the products of decomposition which give rise to the body odor, and that the object of ventilation was the removal of these poisons by dilution with fresh air. To Pettenkofer, carbon dioxide was of significance as an indirect measure of the presence of morbific matter, and it is to him that we owe the assumption that air containing less than a certain amount of carbon dioxide is good and air containing more than a certain amount is bad, because such an amount of carbon dioxide implies the presence of the hypothetical organic poisons.

The theory that miasmatic vapors of the atmosphere were the chief causes of epidemic disease dates back to the days when Hippocrates lighted fires in the streets of Athens to purify the air and put a stop to the plague. This view persisted as a dominant factor

in medical teaching for fifteen hundred years, and when the medical faculty of Paris reported on the epidemic of plague which ravaged the city in 1348, they attributed the disease, as Hippocrates would have done, primarily to malign conjunctions of the planets and, secondarily and immediately, to a corrupted atmosphere. The development of bacteriology gradually, however, dispelled the miasms—which certainly existed as corrupting influences in the thoughts of men, even if they did not affect their physical health. We know today that the communicable diseases are no more influenced by atmospheric vapors than by the conjunctions of the stars. The attempts of Brown-Séquard, Weichardt and others to demonstrate the presence of organic poisons in expired air have been uniformly negatived by the more careful work of subsequent investigators. The recent studies of the New York State Commission on Ventilation indeed indicated that the stale air of an occupied room even when artificially controlled from the standpoint of temperature and humidity, but containing a very high concentration of the chemical products associated with the "body odor," did have two slight but distinctly measurable effects upon human behavior. Subjects exposed to such stale air showed a definite disinclination to physical activity, accomplishing 9 per cent less work in stale than in fresh air; and they showed a marked diminution in appetite, the amount of food eaten at luncheon served in the stale air being from 4 to 14 per cent less than in fresh air of the same temperature and humidity. Experiments in my laboratories at Yale have similarly shown that the growth of young guinea-pigs is quite definitely retarded by exposure to the odors from decomposing organic matter. These effects are, however, only manifest with a concentration of some 20 parts per 10,000 of carbon dioxide (corresponding to an air change of 11 cu.m. per person per hour), and, even then, the effects produced are of relatively minor importance.

The great majority of physiologists now accept the view that the harmful effects of bad ventilation (apart from the presence of industrial poisons and industrial dusts) are no more the result of hypothetical organic poisons than of changes in concentration of oxygen and carbon dioxide. They are due to the other two effects of human occupancy, increase in temperature and moisture, combined with lack of air movement—conditions which, in combination, produce a serious interference with the heat-regulating mechanism of the body. As far back as 1883, Hermans, of Amsterdam, arrived

at the conclusion that the harmful effects of bad air were due, not to its chemical but to its physical properties, and in 1905 Flügge and his associates at Breslau definitely and clearly formulated the modern conception of ventilation—a conception expressed by Prof. F. S. Lee in the succinct formula that the problem is essentially physical, not chemical, cutaneous, not respiratory. The conclusions of Flügge have been amply and universally confirmed by Hill and Haldane in England and by Benedict and the New York State Commission on Ventilation in the United States.

In one form or another these various investigators have repeated a single crucial experiment which may be schematically described as follows. A group of subjects is placed in a closed experimental chamber and kept there while oxygen decreases and carbon dioxide, organic matter, temperature, and humidity increase. They manifest the usual symptoms characteristic of "vitiated" air—headache, general discomfort, lassitude, changes in blood-pressure, pulse-rate, and the like. The attempt is then made to relieve these symptoms in two alternative ways, by permitting the subjects to breathe chemically pure air (admitted for example through a tube from outside) or by lowering the effective temperature of the chamber by cooling coils or the operation of fans, without altering its chemical composition. The first procedure is ineffective, the second completely effective.

It is today clear that the effects of the atmosphere of an ordinary ill-ventilated room are primarily and fundamentally due to a high temperature (combined with lack of air movement and sometimes with high humidity) and to consequent interference with the processes of body temperature regulation. The human body produces by its vital activities from 100 to 400 calories of heat per hour, depending upon the extent of physical exertion. This heat is given off to the surrounding air at a rate which would normally vary very widely with the temperature of the atmosphere. Yet, by one of the most complex regulative mechanisms of the human body, these processes are so beautifully balanced that the actual body temperature does not vary in health more than a degree on either side of the normal (37° C.).

At low temperatures this regulation is in part, but only in small part, accomplished by an increase in heat production; at high atmospheric temperatures there is no such adaptation, the heat production within the body being actually though slightly increased.

It is, then, the rate of heat-loss which varies and which is the chief factor in maintaining the body temperature at a fixed level. When we are in a hot room, the tiny bloodvessels of the skin expand (the face becomes flushed as we say) and bring a large amount of blood to the surface of the body to be cooled off. When we pass into cold outside air, the skin bloodvessels contract, the blood is sent into the inner organs and the cooling effect of the air upon it is thus correspondingly reduced. This is the chief regulatory process which operates at atmospheric temperature below 10° C. As the temperature of the air rises a new factor enters: the increased production of sweat, which is evaporated (if the air be not too humid), with the absorption of heat which always accompanies the change of a liquid to a gaseous form.

The primary effect of high atmospheric temperature is, then, to dilate the tiny bloodvessels of the skin and to concentrate the bloodflow in them at the expense of the inner parts of the body. In part, the feeling of discomfort and lassitude experienced is probably due directly to anemia of the brain and other internal organs consequent on this pooling of the blood in the capillaries. Furthermore, as will be pointed out below, habitual exposure to such conditions leads to a lowered tone of the whole heat-regulating mechanism and an inability to respond to the demands which may be put on it, and in this way exerts a profound and important influence upon susceptibility to respiratory infections.

At still higher temperatures symptoms, of course, become much more acute. The United States Bureau of Mines, in an extensive series of studies recently carried out at Pittsburgh, Pa., equipped a carefully controlled experimental chamber in which they placed subjects, variously clothed, under a very wide range of temperatures, humidities, and degrees of air movement and prolonged exposure to high temperature to the point at which a longer stay appeared definitely dangerous. Restlessness, irritability, headache, and palpitation of the heart, soreness of the eyes, a sensation of oppression on the chest, dizziness, and confusion were the chief symptoms experienced; and weakness and a dragged-out feeling persisted for some time after such experiments had closed. The extreme dry-bulb temperature which could be endured was 69° C. (wet-bulb temperature 38° C., relative humidity 15 per cent) for forty-five minutes while the extreme wet-bulb temperature was 44° C. for thirty-five minutes. The body temperature under such conditions

rose several degrees. The pulse-rate seemed to be the best measure of discomfort, 135 beats per minute corresponding to marked discomfort, and conditions becoming almost unbearable when the pulse-rate exceeded 150 beats. With moderate overheating, both systolic and diastolic blood-pressures fall; under more extreme conditions systolic blood-pressure increases while the diastolic pressure continues to decrease. One of the most important contributions of these investigators is the recent report that basal metabolism (the rate of oxidation within the body) decreases with increasing temperature up to about 26° C. (with 100 per cent relative humidity) and then increases, the rise becoming very rapid when the atmospheric temperature is higher than that of the body. Such a temperature of 26° C. with 100 per cent relative humidity (corresponding in its physiological effects to about 31° C. with 50 per cent relative humidity in still air or 34° C. with 20 per cent relative humidity in still air) is clearly a critical temperature beyond which serious harmful results may be expected. Ideal conditions were found to lie between 17° and 21° C.

The temperature of the air is only one of three distinct atmospheric conditions affecting heat-loss from the body. Humidity and air movement must always be kept in mind as also factors in the problem.

The influence of atmospheric humidity upon heat-loss from the body is quite different at different temperatures. If the air temperature be low, there will be little or no discharge of perspiration and the body will lose heat chiefly by direct conduction and radiation to the surrounding atmosphere. Moisture in the air tends to produce a deposit of moisture on the clothing, and moist clothing conducts heat more readily than dry clothing. Hence, very moist cold air seems chillier than dry cold air. This condition obtains up to a dry-bulb temperature of about 4° to 10° C. Above 10° C. the secretion of sweat begins and the cooling effect of the evaporation of this sweat becomes more and more important as a factor in heat-loss the higher the temperature. Under these conditions the cooling influence of conducting moisture is outbalanced by the fact that moisture in the air interferes with the evaporation of perspiration. Because of this interference with evaporation moist, warm air is far more uncomfortable than dry air at the same temperature. Air containing only 20 per cent relative humidity at 21° C. is about as comfortable (according to the Pittsburgh figures) as saturated air at 18° C.

While hot humid air is recognized as harmful by all physiologists, it has also been claimed that very dry air is also harmful *per se*, favoring abnormal conditions of adenoids and tonsils and causing nervousness and general bodily discomfort. My colleague Ellsworth Huntington has indeed demonstrated that a climate having a mean relative humidity of about 80 per cent for the twenty-four hours is more healthful than one that is more moist or more dry. The New York Commission studies have, however, failed to show any appreciable difference in the health or the efficiency of children in specially humidified schoolrooms as compared with those in rooms where humidity was not controlled.

The third factor in determining the physiological effect of a given atmospheric condition is the extent to which conduction, or evaporation, or both, are favored by the movement of the air. In a still atmosphere the body is surrounded by a layer of air which has been rendered hot and moist by the heat and moisture given off and it is this hot moist air to which the surface of the body is actually exposed.

The Pittsburgh studies indicate that the conditions tabulated below are roughly equivalent to each other from the standpoint of human health and comfort.

Summary of Atmospheric Conditions Producing an Equal Cooling Effect upon the Clothed Body

Dry-bulb Temperatures (Centigrade)

Relative humidity.	Rate of air movement, per minute.			
	None.	30 m.	100 m.	230 m.
20 per cent	20.3	21.4	23.6	25.3
50 per cent	19.2	20.3	22.5	24.2
100 per cent	17.2	18.6	20.8	22.8

Relative humidity is measured by the wet-bulb thermometer and the cooling effect of air movement is estimated by the kata-thermometer. Except where humidity is produced by special industrial processes or where air movement is greatly increased by artificial ventilation, the ordinary thermometer is a reasonably close measurer of indoor atmospheric conditions.

The relatively slight degrees of overheating which obtain in the air of an ordinary badly-ventilated room have three distinct types of influence upon human health and efficiency. In the first place, even a very moderate increase above the optimum of 20° C. leads

to slight but definitely measurable physiological reactions. The body temperature rises while the heart-rate and the respiration increase. In the studies of the New York State Commission on Ventilation it was found in comparing a room temperature of 24° with one of 20° C. (50 per cent relative humidity in each case) that the average rectal body temperature was increased from 36.7° to 36.9° C., the average reclining heart-rate from 66 to 81 beats per minute, the average respiration rate from 17.9 to 19.3.

The most marked effect was that recorded by the Crampton index, an arbitrary measure of the general efficiency of the circulatory system, derived from a comparison of blood-pressure and pulse-rate in the reclining and standing positions. A high Crampton index means that on standing up after lying down the circulatory system is able to increase the blood-pressure so as to meet the greater burden placed upon the system by erect posture without any marked increase in heart-rate. A low index means that on assuming an erect position the heart beats faster and that even then the blood-pressure falls. It is a measure, in other words, of the efficiency with which the circulation responds to a demand upon its power of adjustment. At 20° C. the general average Crampton index was 60; at 24° C. it was 45; and at 30° C. it was 34.

In the second place, under conditions of even slight overheating the performance of physical work is markedly affected. In the New York State Commission studies, 28 per cent less work was accomplished at 30° C. as compared with 20° C. even under conditions of maximum effort, and in a test where the subjects were influenced only by a small bonus, a temperature of 24° C. showed 15 per cent less work performed than was accomplished at 20° C., a conclusion of the most far-reaching importance as an argument for the regulation of temperature in the workshop and factory.

In actual practice, Vernon in England has shown that factory production varies quite regularly with the seasonal temperature and, furthermore, that this variation is relatively slight where a good system of ventilation is in force and relatively great where ventilation is least efficient. Watson finds that in weaving sheds production begins to fall off and fatigue effects are manifest as soon as the temperature exceeds 24° C. with saturated air. (The more carefully controlled laboratory experiments of the New York Commission as we have seen disclosed such an influence at 24° C. with only 50 per cent relative humidity.)

Finally, the work of Hill in England and of the New York State Commission in America has indicated profound effects of only slight degrees of overheating upon the prevalence of respiratory disease. Both English and American observers find the explanation of this effect in observable changes in the membranes of the nose and throat due to overheating and in consequent inability to resist the change from heat to cold on passing into the outdoor air.

One is inclined to ask, if overheating is so serious a factor, why we do not all die in summer. Some of the more sensitive of us do, as evidenced by the high infant mortality in the hot season. There are, however, three very good reasons why high summer temperatures are not so harmful as much less extreme temperatures maintained indoors in winter. One of these reasons is that the air in summer, either outdoors or indoors with windows open, is almost always in active movement. In a series of observations made outdoors in New England summer weather I have found that temperatures at least as high as 24° C. are entirely comfortable and show excellent katathermometer readings. Similarly, a temperature up to 27° C. may be made satisfactory by the use of an electric fan. The cooling effect of air movement is, of course, enormously influenced by clothing. In the Pittsburgh studies air movement produced astonishing results in rendering high temperatures bearable for men unclothed above the waist.

In the second place, the effects of summer temperature are greatly minimized by the factor of acclimatization. We are all familiar with the lassitude which overcomes us on the first warm days of spring; but, as the season proceeds, the circulatory system gradually adjusts itself so as to cope more and more successfully with high atmospheric temperature. Vernon has studied this problem experimentally and finds that during one month of experimentation the endurable limit of wet-bulb temperature for his subjects was increased from 28° to 31° C. The subjects not only showed greater power of endurance but the effect of a given temperature upon their pulse-rate and respiration was markedly diminished.

Finally, and most important of all, the gravest effects, those on susceptibility to respiratory disease, are naturally not produced in summer because they depend on the shock incident upon exposure to the chill outside air following upon the lowered circulatory tone due to a high indoor temperature.

We may summarize our discussion of the physiology of ventilation

as follows: The chemical vitiation of the air of an occupied room (unless poisons or dusts from industrial processes or defective heating appliances are involved) is of relatively slight importance. The organic substances present, manifest as body odors, may exert a depressing effect upon inclination to work and upon appetite; therefore, occupied rooms should be free from body odors which are obvious to anyone entering from without. (Such odors are never perceived by those who have been continuously in the room while they have been accumulating.) Objectionable effects of this sort have only been demonstrated however, with a carbon-dioxide content of over 0.2 per cent, which would correspond to an air change of some 10 cu.m. per person per hour.

The harmful effects of the air of an ordinary badly ventilated room are due to interference with heat-loss from the surfaces of the body. Such effects are very serious indeed. A temperature in excess of 20° C. with little air movement and moderate humidity exerts direct and important effects upon the circulation, causing particularly an increase in body temperature, a rise in heart-rate and a fall in vasomotor tone; it profoundly limits efficiency by causing a disinclination to physical exertion; and it markedly increases susceptibility to respiratory disease.

From a purely physical standpoint, the same rate of heat-loss may be obtained by widely different combinations of the three atmospheric variables, temperature, humidity and air movement. Thus, from the table presented above it appears that a temperature of 17.2° C. with no air movement and 100 per cent relative humidity should produce the same cooling effect as a temperature of 25.3° C. with an air movement at a rate of 230 meters per minute and a relative humidity of 20 per cent.

From the experience of the New York State Commission on Ventilation, however, it appears that high temperatures combined with high-air velocities are actually less desirable than lower temperatures combined with lower velocities, so far as indoor conditions during the winter months are concerned. The New York studies indicate that a temperature of 18.9° to 19.4° C. with about 50 per cent relative humidity and without too much air movement is the ideal one for schoolroom or office ventilation while a still lower figure should be set for work places where vigorous physical work is in progress. For invalids and for the aged, whose powers of heat production have become restricted, considerably higher temperatures must be maintained.

It should be understood that the standards here set are based upon American conditions, with which alone I am completely familiar. Habit and differences in clothing have brought about wide differences in reaction to atmospheric temperatures. In the United States we are forced constantly to fight against our vicious national habit of overheating. In other countries, where such a habit has not existed, the optimum temperature may well be set lower than 19° C. On the other hand I strongly suspect that in England atmospheric temperatures indoors are maintained at too low a level. The death-rate from pneumonia is much lower in England than in America, the death-rate from bronchitis much higher. I doubt the assumption that this is a statistical fallacy and am inclined to believe it to be a real difference due to the well-known clinical difference in the response of these two types of respiratory disease to atmospheric conditions. I suspect that if American houses were kept at 18° instead of above 21° C. and English houses at 18° instead of below 16° C., there would be less pneumonia in the United States and less bronchitis in England.

Where an unduly high temperature exists, the first thing we look to in considering its cause is the question of artificial heat sources. It is very frequently the case that overheating is due, not primarily to lack of ventilation, but merely to the fact that radiators or registers are pouring too much heat into the room. Anyone who has an opportunity to study a wide variety of ventilation problems will recall numerous instances in which regulation of artificial heating would solve the major problems involved, particularly in the case of the living room or the small office.

When a number of people are crowded together, however, a new problem arises, distinct from that of the control of artificial heat sources—the problem of getting rid of the heat produced by the vital processes of the body. We have seen that the average human being gives off about 100 calories of heat per hour. An occupied room will, therefore, become progressively warmer all the time (except in cold weather when heat losses through walls and ceiling are considerable). The primary object of ventilation is to balance this bodily heat production by removing the heated air and replacing it by air which is cool, but not so cold as to produce objectionable and dangerous drafts.

Even in closed rooms the change of air produced by natural ventilation may be surprisingly large. Leakage through walls and

ceilings of a tightly closed room may completely change the air in four or five hours and in rooms with fireplaces in one or two hours. With windows open, the ventilation may, of course, be increased to almost any degree, depending on variations of temperature and wind pressure.

In dwelling houses, where there is no overcrowding, natural ventilation of this kind is generally sufficient for all practical purposes. The hospital ward, too, can, as a rule, be kept in good condition by ordinary window ventilation. The patients are, for the most part, in bed or, at least, warmly clad, and under control; overcrowding is not great, and intelligent supervision of windows is easily possible.

In the use of natural ventilation by means of windows, it is, however, essential to maintain more or less constant watchfulness and to make ample use of the somewhat uncommon attribute of common sense. Many people seem to think that if a single window be opened, either top or bottom, fresh air will come in; but this is by no means necessarily the case. Air does not move of its own volition. It moves as a result either of mechanical pressure or of temperature differences; and air will not enter a room unless a corresponding amount can find an exit at some other point. If a room be otherwise tightly closed, therefore, the opening of a single window may lead to no air change at all. Or the opening of a window may provide an outlet instead of an inlet, as in the case of a hotel room with a transom over the door and a single open window. In such a room hot air from the corridor may flow in through the transom and out through the window all night with little beneficial effect upon the atmosphere of the sleeping room; while closing the transom and opening the window top and bottom would quickly produce the desired result. What one must do in a given case is to give a moment's thought to temperature conditions and wind movement and provide an outlet for warm air above and an inlet for cool air below.

In schools, in crowded offices and workrooms and in auditoria, such natural ventilation by the use of windows alone is not likely to prove satisfactory. When the air outside is nearly as warm as that indoors, the temperature differences, unless a strong wind is blowing, will provide an altogether insufficient supply of air to remove either body odors or excess heat produced in a crowded room. In cold weather, on the other hand, insuperable difficulties

arise as a result of inequalities of distribution. In a school or a factory where there is a row of 5 or 10 or 20 persons extending from a window across to the inner wall of the room, it is out of the question to ventilate by admitting the untempered air of winter. Either the temperature near the window will fall so low as to chill those in that vicinity or the temperature at the other side of the room will rise too high, the latter result being the more usual one.

Natural ventilation, then, will generally serve for the living room and for the hospital ward, but not, as a rule, and without certain modifications, for the school, office, factory, and auditorium. In such places special provision must be made for the removal of heated air and the supply of fresh cool air. There are two general methods of attaining this end: gravity ventilation and fan ventilation; but, before describing these processes in detail, it will be desirable to review for a moment the development of popular conceptions of good ventilation and the embodiment of these conceptions in legislative standards.

The system of gravity ventilation—in which inlet and outlet openings are provided but reliance for the actual movement of the air is placed upon temperature differences are "chimney action"—was the one used by the pioneers of ventilation practice, Tredgold and Reid, in the first three-quarters of the nineteenth century. With the acceptance of the Pettenkofer carbon dioxide test for alleged organic poisons there grew up a desire for a more rapid change of air than the gravity system could provide and the progress made by engineers in the construction of fans led to the development of systems in which these centrifugal fans were used for the mechanical propulsion of larger volumes of air.

According to the Pettenkofer conception a concentration of carbon dioxide in excess of 6 parts per 10,000 was supposed to be associated with a dangerous proportion of poisons in the air.

On the assumption of such a carbon dioxide standard it is, of course, very simple to calculate a standard of per capita air supply. Since we know how much carbon dioxide is eliminated by an average individual per hour, the dilution of this carbon dioxide by outside air containing 3 parts per 10,000 so as to keep the room air below a given concentration is merely a matter of arithmetic.

The average individual produces approximately 0.017 cu.m. of carbon dioxide per hour in the process of respiration. Normal outdoor air contains 0.0003 part of carbon dioxide. If we assume that

0.0006 part of carbon dioxide represents an upper permissible limit (more carbon dioxide than this being held to indicate a dangerous concentration of hypothetical body poison) it is easy to compute the amount of fresh air (containing 0.0003 part CO_2) which must be brought in per person per hour to prevent the concentration of CO_2 in the room air from rising above 0.0006 part. This we do by the formula

$$\frac{0.017}{0.0006 \text{ to } 0.0003} = x$$

which gives us 57 cu.m. per hour as the desirable per capita air supply per person. If we accept such a standard, and if we further assume that the air in a room can be changed from four to six times an hour without producing objectionable drafts, we arrive at the conclusion that each individual in an occupied room must be allotted a cubic air space of 10 to 15 cu.m.

Thus, in regulations for the control of factories and workshops, Austria demands a cubic capacity of 10 cu.m. and a surface space of 2 m. which are subject to increase should certain circumstances render it necessary (dust, fumes, etc.); Belgium also demands 10 cu.m. per worker and a renewal of 30 cu.m. at least per hour and of 60 cu.m. in specially unhealthy workshops; Canada requires 11 cu.m. per worker; Denmark 8 cu.m. (which the Factory Inspection Department may increase to 12); France demands 7 cu.m. at least and 10 cu.m. in certain industries (laboratories, kitchens, stores, shops and public offices). The air of workshops shall be renewed in such a way as to maintain the standard of purity essential to the health of the workers (Decree of July 10, 1913), and the general ventilation for any enclosed space shall be such as to prevent an undue increase of temperature.

In Great Britain 7 cu.m. is demanded as a minimum; further the Cotton Factory Act and other Orders impose a maximum of 9/10,000 CO_2 for the textile industries with a cubic capacity of 11 m. when overtime is worked unless electric light is installed. In Italy the draft regulations for industrial hygiene provide for a uniform cubic capacity of not less than 10 m. per person as well as a minimum surface space of 2.5 m. Air in closed workrooms shall be frequently renewed so that the CO_2 rate produced by exhalation shall not exceed 10 in 10,000. Where this rate is exceeded factory inspectors shall have authority to demand that natural ventilation be com-

pleted by artificial ventilation. Whatever the system of ventilation, all drafts liable to inconvenience workers engaged at a fixed point and exceeding a rate of 0.5 m. per second must be avoided. In Holland, good natural ventilation must be provided where mechanical ventilation is not installed. Natural ventilation is deemed insufficient where the total surface of apertures communicating directly with the outer air is not at least 5 cu.dc. per person and at least 40 cu.dc. exclusive of doors. The third part at least of the surface of such apertures must be situated at a minimum of 1.8 m. above the ground and it ought to open independently of all other apertures demanded by legal requirements. Sweden demands 10 cu.m. and Switzerland, likewise, with a minimum height for workrooms of 3 m., etc.

A reasonably ample per capita air space is desirable from many standpoints; but the rigid application of the Pettenkofer formula in regard to per capita air supply involves needless expense and, as I shall point out, may involve real damage to health as well. In Europe this type of ventilation standard has generally been applied with reasonable discretion. In Sweden, France and Germany, for example, 12 to 22 cu.m. of air supply per child are required in the schoolroom. In the United States, on the other hand, the sincere but misguided efforts of our ventilating engineers have succeeded in writing into the laws or official regulations of many of our states the requirement of an air supply of 51 cu.m. per child per hour—a standard which absolutely requires fan ventilation; and we are now engaged in a vigorous effort to obtain the repeal of regulations of this type.

All of these legislative standards in the light of more modern views as to the objectives of ventilation now appear to be more or less obsolete, or at least very incomplete. We conceive ventilation today primarily as a procedure for removing the excess heat produced by the human body and replacing it by fresh air that is cool but not too cold. The amount of air needed for this purpose will obviously bear no necessary and direct relation to the number of persons in a given confined space unless we also take into account the opportunity for direct heat-loss through windows, walls and ceiling. Under certain conditions, as for example in an interior auditorium with no appreciable heat-loss, the standard of 50 cu.m. per hour (legally required for schools in many American states) will prove substantially correct. An average adult gives off approx-

imately 100 calories of heat per hour, and this is almost exactly the amount of heat necessary to raise the temperature of 50 cu.m. of air from 16° to 21° C. In factory workrooms where there are special heat sources such as forges, furnaces and annealing ovens, the amount of air necessary to maintain good ventilation may be materially more than this. In certain annealing shops for example, 30 changes of air per hour have been effected with notably successful results. On the other hand, where the room is relatively small and has one or more outside walls the necessary air change may be materially less.

In the studies of the New York Commission on Ventilation it was made clear that gravity ventilation, with an air change to 20 to 30 cu.m. per hour possessed certain distinct advantages, as compared with the more costly process of fan ventilation. The system of gravity ventilation studied involved three distinct elements. Air was admitted at the windows over slanting plates (usually of glass) which served as deflectors to direct the incoming air upward and avoid direct drafts upon the pupils sitting near by. In the second place a radiator was placed under each window to temper the incoming air; and finally an exhaust duct was provided, opening from near the ceiling on the inner side of the room and extending up to the roof, for the escape of the hot vitiated air. No fans need be provided but it is often valuable to place a heating coil in the exhaust duct to emphasize its chimney action.

Such a system, of course, gives materially less air change than the ordinary fan-ventilation system as used in the United States (perhaps 20 to 30 cu.m. instead of 50) but this air change is quite adequate to avoid objectionable odors. The second difference, correlated closely with the first, is to be found in the fact that window-ventilated rooms are characteristically maintained at a temperature slightly lower than fan-ventilated rooms. In a window-ventilated room with a comparatively slight air movement, temperatures between 19° and 20° C. were borne with comfort; while in a fan-ventilated room with the increased heat-loss due to greater air movement the temperature had to be maintained between 20° and 21° C. Even under such conditions the rate of heat-loss as measured by the katathermometer is greater in the fan-ventilated room, but our results indicate that the katathermometer somewhat exaggerates the effect of air movement upon the clothed body.

In general our studies showed that the cooler rooms with the

lower air-flow were distinctly more comfortable than those with a higher temperature and higher rate of air change. Furthermore, a marked effect of overheating and overairing upon respiratory disease was observed in comparing these two types of ventilation. The classrooms in this study were divided into three groups: *A*, Rooms ventilated by window inlets and gravity exhaust and kept at a mean temperature of 15° C.; *B*, rooms ventilated by window inlets and gravity exhaust and kept at a mean temperature of 19.1° C.; *C*, rooms ventilated by fan supply and gravity exhaust kept at a mean temperature of 20.3° C. Typical carbon dioxide records in these schools showed an average of 7.7 parts in rooms of Type *B* and 5.9 parts in rooms of Type *C*, corresponding (according to the more exhaustive data from other series of experiments) to approximately 34 and 51 cu.m. of air supply per hour per pupil respectively. The fundamental difference between the three types of rooms was that in Type *A* we were using a low flow of cold air, in Type *B* a low flow of cool air, and in Type *C* a high flow of warm air. The overheating in the rooms of the last type was not extreme (mean 20.3° C.) and appeared to be entirely inseparable from the high air flow. With an air supply of 51 cu.m. per hour it is essential to maintain temperatures generally over 20° C. to avoid unpleasant drafts; while with a lower air supply temperatures can be kept well below that figure.

When the records for the two years were analyzed and averaged, it appeared that the amount of respiratory sickness in the 15° rooms and the 19.1° C. rooms was essentially the same; while the 20.3° C. rooms showed an 18 per cent excess of absence due to respiratory sickness and a 70 per cent excess of respiratory sickness among pupils in attendance. After a most careful study of social and economic conditions and of all other possible factors bearing on the case, there seemed no escape from the conclusion that even the slight degree of overheating combined with draft had exerted a very definitely harmful influence upon the susceptibility of the children to respiratory infections.

For the ordinary schoolroom, therefore, we believe that the system of window-gravity ventilation is not only more economical but distinctly more healthful than the system of fan ventilation. In a large or crowded office or workroom, however (particularly if special heat sources be present) and in the auditorium, natural heat-loss through walls and ceiling will be relatively small and the difficulty

of admitting outdoor air from windows without draft will be materially increased. Such occupied spaces cannot, as a rule, be satisfactorily ventilated by a window-gravity system and we must here resort to fan ventilation.

Two general types of fans are used in securing forced ventilation, the propeller fan and the centrifugal fan. The propeller type operates on the principle of the ordinary electric desk fan, having curved vanes which drive the air at right angles to the general plane in which the fan itself revolves. This form of fan will move considerable volumes of air but only against low pressures, and is well adapted to supplement natural ventilation when only a moderate increase in air change is essential. Propeller fans are often placed in the outer walls of workrooms for example, or over kitchen ranges, discharging outward, to accelerate natural agencies in the exhaustion of vitiated air. They are not, however, suited for use with duct systems of ventilation where the air must be moved against considerable pressure. When the pressure rises, their efficiency falls off and a back current is often created, the air flowing in one direction at the periphery and in the opposite direction near the center of the fan.

For systems of duct ventilation the centrifugal type of blower fan should generally be used. In such a fan air is drawn in by lateral openings at the center of the plane of revolution and forced out through a tangential duct by the action of a series of parallel blades. Fans of this pattern may be used as pressure or plenum fans when they deliver to the ventilating ducts and draw in their supply freely from an air chamber surrounding them. Or they may be used as exhaust fans if the central opening is connected to the ventilating ducts and the delivery is to the outer air. In some buildings with elaborate plans for ventilation both plenum and exhaust fans and ducts are provided.

Plenum ventilation is often combined with air washing or artificial humidification. In such a system the incoming air is first heated to a moderate degree (10° to 20° C., depending on various conditions) by passing through the tempering coils, which are lines of steam piping like those in an ordinary radiator, but so constructed as to present the largest possible surface to the rapidly moving air. From the tempering coils the warmed air passes through the spray chamber or humidifier. This is a chamber filled with fine mist of water produced by some form of spray discharge, and at its outlet

end the air passes between a series of overlapping eliminator plates or baffles which change its direction suddenly many times. Contact with these baffle plates removes the excess of moisture which drains off from the eliminator to a collecting pan below.

Three things occur in the spray chamber, when it operates sucsuccessfully. The air is humidified nearly to the point of saturation. It is cooled by the loss of the amount of heat required to transform the water taken up from the liquid to the gaseous form (3° to 5° C. in summertime), and it is washed free from a portion of its suspended dust particles.

Finally, on leaving the spray chamber the air passes through a second set of heating coils, where its temperature is brought up to the final point desired, ranging from perhaps 15° C. where the removal of the heat produced in an auditorium is necessary to 65° C., where a large amount of indirect heating must be accomplished.

The temperature of the air delivered by the fan may be regulated automatically by thermostats controlling either sections of the heating coils, or dampers which admit a varying proportion of by-passed air, not exposed to the heating coils at all. The humidity of the air delivered can be regulated by controlling the relative temperatures of the tempering and the final heating coils since the air passing the humidifier is supposed to be saturated at the temperature at which it leaves the spray chamber and its final humidity will obviously depend on the increase in temperature to which it is subjected after this point.

From the fan chamber, the tempered air, whether it is humidified or not, is delivered to the various parts of the building by a system of ducts carried up, though, or along the walls. It may be pointed out that many systems of fan ventilation in actual use fail to give satisfactory results because the ducts are so imperfectly proportioned as to distribute the air unevenly between the different parts of the building. Thus, in one office building studied by the writer an exhaust system was installed which gave reasonably good results on the six lower floors, while on the upper floors there was either no air-flow at all or vitiated air from the lower part of the building was being forced into these rooms through the supposed exhaust ducts. The problem of the proper portioning of air ducts is by no means a simple one, as emphasized particularly by the studies presented in the second report of the British Departmental Committee

on the Ventilation of Factories and Workshops (1907). It is important that individual duct dampers should be provided so that the air supply to each room can be independently governed; and by somewhat elaborate but wholly practical systems of double or individual duct ventilation even the temperature of each room can be separately controlled to meet varying conditions of weather and occupancy. Where air is delivered to a large factory workroom by a long duct running along the ceiling, it is common practice to construct the duct of a tapering form, becoming gradually smaller from the inlet to the outlet end. Recent studies have shown that a uniform straight duct is likely to give quite as good results; but that the main duct should discharge into the room not by mere lateral openings but by short branch ducts slanted forward in the direction of the air current.

The linear velocity of air maintained in systems of fan ventilation will usually vary from 480 to 600 m. per minute in the main duct near the fan. As the air passes through horizontal branch ducts and vertical stacks its velocity is gradually reduced and at room inlets it is usually between 90 and 240 m. per minute. It is desirable that it should not exceed 90 m. per minute at such points in order to avoid unpleasant drafts. If, then, it is desired to provide an air supply of 50 cu.m. per hour, the inlet registers in any room should have a total area equal to 1 sq. dcm. per capita.

In addition to providing a given total amount of air to the room to be ventilated, considered as a unit, it is important to secure a reasonably equable distribution of air within the different parts of the room itself. Local temperature differences and other factors which affect this distribution are complex and variable. It is by no means rare to observe grotesque mistakes in actual practice such, for example, as an exhaust fan of the propeller type placed over an open window producing a strong local up-current with no material benefit to the room as a whole.

There are two general systems of room air distribution in common use; the upward and the downward system. The latter plan, which is usually employed in schoolrooms, ventilated on the fan system, brings the fresh air in at the top of the inner or warm wall of the room and takes out the vitiated air at the bottom of the same wall. It is assumed, in such a system, that heating is combined with ventilation and that the incoming air will be warmer than the general room air. In such a case the New York State Commission

studies indicated that it was generally best to bring the plenum air supply in on the short wall of the room near the ceiling and to place the outlet near the floor of the same wall. The warm air shoots across the upper part of the room and then, as it cools, passes backward through the zone of occupancy.

If ventilation is frankly recognized as a procedure for supplying fresh, cool air to remove the excess heat produced within a confined space, an upward system of ventilation seems more logical. This plan involves the introduction, at or near the floor, of air cooler than the general air of the room, this air to become gradually warmed as it rises to exhaust outlets at the ceiling. With such a system, however, it is very difficult to avoid unpleasant drafts, and downward ventilation from ceiling inlets is the most satisfactory way of supplying air to a large auditorium.

The problems of ventilation are not nearly so subtle and mysterious as the layman is often inclined to believe. Nine-tenths of them can be solved by the exercise of a little common sense along the following simple lines.

1. The first step is the placing in every living room, schoolroom, office, and workroom of a thermometer and systematic noting of the temperature recorded.

2. If the temperature is found to exceed 20° C., the next step is to attempt a control of overheating by moderating artificial heat sources such as radiators or registers.

3. If overheating still continues, the effort should be made to introduce fresh cool air by adjustment of windows, transoms, and the like, providing an inlet for cool air below and an outlet for warm air above and avoiding unpleasant and dangerous drafts. Success may generally be attained by these simple means in the living room, the hospital ward, and in many offices.

4. In larger offices, in schools, and in many factory workrooms, we must go a step further and provide for a system of window-inlets and gravity-exhaust, such as was found so satisfactory by the New York State Commission on Ventilation. This gravity-window system involves three simple elements; the admission of fresh air over deflecting surfaces such as are provided by slanting window boards or tilting sash windows; the tempering of this incoming air by radiation extending across and below the windows to be used; and the provision of gravity exhaust ducts (with or without heating coils) opening from a point or points near the ceiling on or near

the inner wall of the room. Such a procedure will change the air at a rate of 17 to 25 cu.m. per hour per person, amply sufficient to guard against unpleasant odors. It will maintain a moderate current of cool air, far more pleasing, and very much more healthful, than the heavy stream of warmer air produced by the fan system. It is, in general, the ideal method for the ordinary schoolroom.

5. In schools so situated that dirt, smells, or noise prevent the opening of windows—in factory workrooms which are very large and crowded or which contain special heat sources or sources of dust and fumes—and in large auditoria, gravity ventilation will not usually suffice and we must perforce turn to mechanical ventilation. It is at this point that the exercise of lay common sense gives way to that of expert knowledge; for, if a fan-ventilation system is to be used at all, it is essential for efficiency and economy that it should be designed by an experienced ventilating engineer.

"Finally," to quote the words of the New York State Commission Report, "it must be emphasized in closing this discussion that the avoidance of overheating is the primary essential in all systems of ventilation. Air change, direction of flow, and all other factors are secondary. The most important article of ventilating equipment is the thermometer; and however simple or however complex an apparatus may be installed for air condition, a constant and intelligent vigilance in regard to operating and overheating is the price of health and comfort."

LABORATORY AND CLINICAL METHODS*

By H. R. M. LANDIS, M.D.

I HAVE selected as my text a statement made by Francis Bacon: "Some dispositions evince an unbounded admiration for antiquity, others eagerly embrance novelty; only a few can preserve the just medium, and neither tear up what the ancients have correctly established, nor despise the just innovations of the moderns."

Whether it is true to the extent that many would have us believe, it is certainly true that there are not a few who think that the art of diagnosis has suffered with the introduction of scientific tests performed in the laboratory. Stokes (*Modern Clinical Syphilis*, 1926), for example, states that clinical and laboratory diagnosis in medicine are too often spoken of as maintaining a species of tacit rivalry, not to say antagonism, toward each other. This attitude may be explainable in part to the fact that when the laboratories came along they were captured in an educational sense by clinical medicine, but they have now captured their captor. A more logical explanation is that suggested by Stokes, namely, that it is at all times still difficult for the physician, sharing the common desire of human nature for touchstones and open sesames, to realize that syphilis, or for that matter any disease, will never wholly yield to a single diagnostic key. While no one will deny the positive need for systematic use of certain laboratory tests, amounting in fact to routine application in certain aspects of disease, the diagnostician, as Stokes insists, should never forget that the substitution of a mechanically ordered test for a frame of mind, results in some of the poorest diagnostic work in medicine. For all its value, any procedure which substitutes a test-tube reaction for a primary acuteness of perception debases the clinician and costs him often in the end more efficiency and self-respect than its accuracy and easy accessibility can ever justify. It is this tendency that has led to the loss of respect for clinical as compared with laboratory evidence.

The multiplicity of accessory diagnostic aids has produced the

* Read June 1, 1927.

same confusion that besets modern thought in all fields. As Durant has expressed it: "Our modern danger is that we suffocate with uncoördinated facts; our minds are overwhelmed with sciences breeding and multiplying into specialistic chaos for want of synthetic thought and a modifying philosophy." The modifying philosophy needed in medicine is that physicians cease to blindly enslave themselves still further to any single routine aid. They should be inspired, as Stokes puts it, to recanvass and furbish up their clinical acquaintance with disease. In no other way, I take it, will the two methods be made complementary to each other. It is just as essential to have a proper understanding of the limitations of the accessory diagnostic aids as it is of the shortcomings of clinical evidence.

It is hardly necessary to remind you of the debt we owe to Sir James MacKenzie in advancing our knowledge of cardiac disease. Recalling that he was what is commonly called a "country practitioner," and that much of his work was based on a mechanical aid, it is interesting to note his conception of what clinical medicine should be: "The advances that have been made in special branches, particularly those pursued in laboratories and by the use of mechanical devices, are all reflected in the field of clinical medicine. In an attempt to be up to date, physicians use the methods of these auxiliary branches, so that clinical medicine toils laboriously in their rear, and to a great extent becomes subservient to them. It should be realized, and should be strongly and persistently insisted upon, that all these special methods fall far short of the ideal of what is wanted in clinical medicine, and that the pursuit of clinical medicine involves problems, peculiar to itself, which none of these methods can ever hope to solve. There is not a single mechanical or laboratory method ever introduced but has had an extremely limited sphere of usefulness. Time and again great expectations have been raised on the announcement of some wonderful discovery that was going to have a revolutionary effect upon clinical medicine; but in every case, as time went on, when its sphere of usefulness came to be recognized, it was found to be a very limited one. The impression that the results obtained by a mechanical method are more reliable and scientific than those obtained by the use of the unaided senses, is but a belief based on a false analogy. It is assume that because the experimentalist obtains certain records which are capable of demonstration, these are of more value than the informa-

tion derived by the doctor from questioning of his patient, or by the use of his own senses trained by long experience. Those who reason thus unconsciously compare unequal things. In laboratory experiments it is impossible to obtain any information from the great field of objective impressions. In man this is the most important field, and far outruns the field of mechanical exploitation in giving us information as to the nature of the disease. The laboratory worker obtains his results by a delicate mechanical contrivance. The physician has to train his senses, and this can only be done by a long process of education only capable of being acquired by the constant contact with the patient."

In speaking of the early physicians in India in the eighteenth century, Sir Leonard Rogers pays the following tribute to their powers of acute observation: "That writers who lived before the days of modern microscopes, or even temperature charts, should have been able to classify and describe the fevers of India to the extent they did, teaches a valuable lesson in these days when clinical studies are at a discount." It is also of interest to those of us who are but slightly familiar with tropical diseases and which most of us regard as curiosities, to read in his introduction to *Fevers in the Tropics* that Rogers "has been led by his researches to conclude that a large proportion of fevers in the tropics can be diagnosed within two or three days by purely clinical methods. It will thus be possible in the remaining doubtful cases for a microscopical examination of the blood to be made as a matter of routine, and a clear idea to be got of all fever cases, whereas, under ordinary conditions of work in the tropics time does not permit of the use of the microscope in every case." This it seems to me is the crux of the situation.

The great majority of physicians, particularly those practising in small towns and rural communities, are for the most part unable to avail themselves of many of the modern scientific aids to diagnosis. We all realize the value of many of these accessory aids, and we take it as a matter of course that a well-equipped metropolitan hospital makes use of the electrocardiograph, for example, in further elucidating the cardiac arhythmias. Is this, however, necessary as a routine measure? According to Sir Thomas Lewis, it is not. As he has shown, careful attention to the clinical phenomena makes it quite possible to recognize all but a small proportion of these cases.

Diagnostic problems, for the most part, fall into three groups: There is, for example, the group in which a correct solution is

dependent entirely on the presence or one or more of the special tests. This group is, I believe, much smaller than most physicians think. In many cases the recognition of a diseased condition is credited to a laboratory test, when as a matter of fact it could have been detected by clinical methods had those been used intelligently. Thoracic aneurysm occurs to my mind as a notable example of how an obvious condition can escape notice as the result of slovenly clinical methods. I have seen patients with thoracic aneurysm who have gone from dispensary to dispensary without their trouble being recognized for the want of a careful glance. I do not wish to be understood as insisting that the methods of physical diagnosis alone will invariably recognize all thoracic aneurysms. I do believe, however, that clinical methods will detect the great majority of such cases, and that at least a suspicion of the existence of the trouble may be obtained in most of the remainder. One can have little sympathy for the humiliation of the clinician who is confronted with an *x*-ray film showing a large tumor mass in a patient in whom he has failed to notice the slight bulging of the sternum, pulsation, the unequal pupils and the dulness on percussion, or to have failed to take heed of the subjective pressure symptoms.

By far the largest group seeking diagnostic aid is that composed of patients in whom the source of their trouble is quickly apparent or is brought to light by careful history-taking and physical examination. I would cite a disease with which I have had some experience, namely, pulmonary tuberculosis. In the great majority of patients suffering from pulmonary tuberculosis, irrespective of the stage, clinical methods, plus examinations of the sputum, serve to recognize the trouble. Making a rough guess, I should say that the man who has a reasonable familiarity with the natural history of this disease, its pathology and the symptoms, should arrive at a correct conclusion in at least 90 per cent of the cases he sees. There will always remain a small group in which every known method will be needed to establish a diagnosis. Of all the accessory aids which have been introduced to facilitate the recognition of pulmonary tuberculosis, the only ones, as Baldwin (*De Lamar Lectures*, 1925–1926) has pointed out, that thus far have any value in diagnosis, are the demonstration of tubercle bacilli, tuberculin tests and the complement-fixation test. In the diagnosis of syphilis and cardiac disorders this same view is held by Stokes and MacKenzie, whom I have already quoted.

Finally, there is that large group which Peabody has recently described. Patients who "have nothing the matter with them." If what may be termed the laboratory group is much smaller than is usually thought, this group is far larger than most physicians appreciate. When I was a medical student, if any one had the temerity to ask what the treatment should be, he was put off with the answer that that was of secondary importance. Once you had the diagnosis, the proper treatment followed as night follows day. In common with many young practitioners, I finally learned that it is quite possible for people to suffer from ill-health of varying degrees of severity, and which in some instances amounts to almost complete disability, without the presence of an organic lesion.

It is in this group that the human element plays such an important part. As Peabody has expressed it, in patients whose symptoms are of functional origin, the whole problem of diagnosis and treatment depends on the physician's insight into the patient's character and personal life, and in every case of organic disease there are complex interactions between the pathologic processes and the intellectual processes which he must appreciate and consider if he would be a wise clinician.

Far too many physicians, once they have eliminated the possibility of organic trouble, lose interest or are apt to assume that inasmuch as no anatomic lesion exists the symptoms will eventually disappear. They fail to appreciate that while the symptoms have no anatomic basis, they are none the less distressing and disturbing, and there is nothing imaginary about them. A pyloric spasm, for example, due to a disturbance of innervation, in my own case always brought on by fatigue, is just as distressing as one due to duodenal ulcer. The value of laboratory methods in this group cannot be denied, for without the assurance that organic disease is not present, one remains more or less in the dark.

DISCUSSION

Dr. Eugene L. Opie: Dr. Landis' quotation from Bacon defines as accurately as possible the relation of the clinic to the laboratory. Both wish to hold good things of the past and adopt new methods as they present themselves. There is a good deal of difficulty in distinguishing the proper

attitude of the clinician from that of the laboratory worker for the spirit underlying both must be identical. The physician who comes into personal contact with his patient must interpret what comes from the laboratory. Information gained by use of the ophthalmoscope or by hydrogen-ion determination are subject to the same fallacies of judgment. Procedures which are new often excite attention out of proportion to their real value, but experience finally decides their worth. Correct judgment in clinical medicine still requires an accurate knowledge of pathological anatomy, for, with etiology, it is the basis of pathogenesis. Roentgenological methods make it possible to apply knowledge of pathological anatomy to the patient during life and in many directions obviously furnish information not obtainable by the usual method of physical diagnosis.

Dr. Baxter L. Crawford: I heartily agree with many of the points Dr. Landis brought out, and there are one or two I would like to lay more emphasis on, for example, the importance of laying emphasis on serological tests. In the hands of skilled workers these tests are very valuable, but not infallible by any means. In the hands of the inexperienced they are unreliable. I think the majority of physicians realize this. The Wassermann reaction, I think, is dependent largely upon the person who does it, and the same is true of many of our bacteriological tests.

Dr. Astley P. C. Ashhurst: I am always glad to agree with Dr. Landis, and especially when he upholds clinical as opposed to what he calls laboratory methods; although, as Dr. Opie points out, the methods are really the same. But I do not think Dr. Landis is quite justified in considering laboratory methods as merely mechanical. Anyone who sends sections of tumors over the country for pathological examination and receives back from the head of each laboratory a different diagnosis will perceive that the diagnosis is not mechanical, but depends upon the eye at the end of the microscope and upon the brain behind that eye.

It is true that many students, especially many undergraduate students, rely too much on the laboratory. This no doubt, is because they receive very little if any clinical instruction until the third or fourth year of their course. When they reach me for clinical work in their fourth year, I try to make them do everything they can with their eyes and fingers and their ears and even with their noses, as well as with their brains, before they are allowed to know what the laboratory tests have shown; and it is surprising how far they can go in that way.

I saw in my ward a few days ago a man, about thirty-two years of age, with a tremendous bilateral hydrocele, which he said came on after a sudden strain about three or four months previously. Now I argued that this was no reasonable thing to occur in a healthy man; he must have had some predisposing disease. Then I remembered that sometimes men who had syphilis had hydrocele and *vice versa*. So I said to my intern, I suspected syphilis as a predisposing cause, and was not at all surprised to be told that the patient acknowledged having had a chancre eight years ago. I did not have to wait for the Wassermann test to make myself sure that this man had

syphilis. And though my intern remained skeptical, and wished me to do a radical operation for hydrocele, and seemed a little peevish when I recommended that the hydrocele be tapped, a quite different expression came over his face when he had drawn the fluid and found both testicles much enlarged; and when the laboratory had reported that the hydrocele fluid gave a "*plus* four" Wassermann reaction, this intern had a much greater respect for my clinical judgment and diagnosis.

So, Mr. President, I believe Dr. Landis has done well to call attention again to the need for reliance on clinical methods, and the propriety of teaching them to our students; always having a reliable laboratory available, at the head of which an eye and a brain will be at work to check our clinical methods, correct our errors, confirm our correct conclusions, and in cases of doubt (both clinical and laboratory doubt) help us to find a path out of our difficulties.

NUTRITION AND TUBERCULOUS INFECTION IN CHILDHOOD*

By H. W. HETHERINGTON, M.B.

Malnutrition and latent tuberculous infection are both subjects which have received gradually increasing attention from the medical profession in recent years.

Pioneer work in malnutrition was begun by Dr. William Emerson, of Boston, in 1908, and from that time on its importance has been slowly recognized by physicians and welfare organizations. Nevertheless, the report of the Surgeon General during the World War, which showed only slightly more than 50 per cent of drafted men to be fit for service in the first line, was startling. The fact that the defects of adults are traceable to defects and habits originating in childhood revealed malnutrition and its causes as one of the most important fields of medicine.

The use of tables showing the average weight of children for height and age is essential to the diagnosis of malnutrition. Some physicians consider 10 per cent underweight or more to constitute malnutrition, while others think this standard to be too low and believe that 7 per cent underweight is the borderline which should be adopted.

The recognition of latent tuberculous lesions is also a question of great importance. The hope that sanatorium, segregation, education and treatment would solve the tuberculosis problem has faded. Tuberculosis is still one of the leading causes of death, and the vast majority of even moderately advanced or minimal cases who receive sanatorium treatment still die of this disease. Prophylaxis rather than attempted cure should be our aim. Latent tuberculous lesions may exist for months or years before they become manifest disease. If we could recognize individuals with such lesions, proper prophylactic measures would usually prevent the development of their disease.

* Read June 1, 1927.

A variety of clinical symptoms and signs have been described in association with the latent or masked form of tuberculous infection which occurs frequently in childhood. Among the symptoms so described are "colds" of head and chest, hoarseness, cough, expectoration, anorexia, fatigue, malnutrition, asthenia, anemia and fever, and among the physical signs parasternal or paravertebral dulness, vesiculobronchial breathing in certain areas and D'Espine's sign.

They may be grouped under four headings:

1. Symptoms of upper respiratory infection, namely, frequent colds, hoarseness and cough, sometimes with expectoration, are evidence, in most instances, of catarrhal inflammation, perhaps spreading from some area of focal infection. They are no more indicative of tuberculosis in the child than in the adult.

2. Physical signs, including D'Espine's sign, paravertebral and parasternal dulness and vesiculobronchial breathing in the interscapular region may be found in normal children. In the report of the study of the chests of normal children delivered to the National Tuberculosis Association, the following opinion is expressed: "D'Espine's sign, as indicative of enlarged tracheobronchial lymph nodes, is, to say the least, of doubtful value. . . . The determination of a diminution of resonance in the interscapular region requires such a nicety of technic that even masters of percussion disagree as to the presence or absence of significant findings in this region of the chest." And finally: "Exaggerated vesiculobronchial breathing, especially well heard in the areas overlying the great bronchi (that is anteriorly, at the level of the first interspace and second rib, just lateral to the sternal margin and posteriorly, especially on the right side at the level of the second to fourth dorsal spine), is often incorrectly interpreted as evidence of pulmonary disease."

3. Fever, indicated by a slight abnormal elevation of temperature, is an uncertain guide in children, for the heat-regulating mechanism is either incompletely developed or extremely sensitive, so that trivial and obscure causes may produce fever.

4. Malnutrition, accompanied by asthenia, anemia and fatigue after slight exertion, is usually caused by insufficient nourishment, poor hygiene and focal chronic infections.

There are two means by which the existence of latent tuberculosis can be clearly shown: (*a*) A positive tuberculin reaction means infection with tubercle bacilli, and a negative tuberculin reaction

with few exceptions, indicates the absence of infection. The occasional failure of the tuberculin reaction in the presence of tuberculosis is of relatively little importance. (*b*) Roentgenological demonstration of caseous or calcified nodules of lungs or tracheobronchial glands is again, with few if any exceptions, evidence of tuberculous infection. These lesions are recognized by their characteristic appearance in *x*-ray films. They should not be confused with the round homogeneous shadows (3 to 5 mm. in diameter), seen in the hilum region in almost every film of the chest and even in films of young infants, for they are bloodvessels axial to the ray. Pathological densities in the lung or tracheobronchial glands are irregular, with granular or mulberrylike appearance, and are visible in films taken at various angles.

During the past two years a tuberculosis survey of the children of three Philadelphia public schools has been undertaken by the Henry Phipps Institute. Approximately 60 per cent of the children examined were colored. Tuberculin tests were made by injection of 0.01 mg. of old tuberculin into the dermis; if the reaction was negative, at the end of forty-eight hours a second injection of 0.1 mg. was made; if the reaction was again negative after forty-eight hours a third injection of 1 mg. was made. The reaction was considered positive if an area of redness 10 mm. or more, accompanied by edema, developed in response to the injection of 1 mg. of old tuberculin. Failure to react thus to 1 mg. was designated negative and considered a lack of sensitivity to tuberculin. The weight of the child was compared with the normal weight as determined by the tables of Dr. Bird T. Baldwin and Dr. Thomas D. Wood.

Nineteen hundred and ninety-nine children between the ages of five to sixteen years inclusive have been examined. They have been divided into (*a*) children who were normal in weight or overweight, and (*b*) children who were underweight. The number and percentage of each group positive and negative to tuberculin is shown in Table I.

TABLE I.

	Nutrition	
	Normal or overweight.	Underweight.
Total number	1270	729
Negative to tuberculin:		
Number	348	208
Per cent	27.4	28.5
Positive to tuberculin:		
Number	922	521
Per cent	72.6	71.5

If latent tuberculosis were a factor in the production of underweight, underweight children would give a larger proportion of positive tuberculin reactions than overweight children. Of the normal or overweight group 72.6 per cent and of the underweight group 71.5 per cent react to tuberculin. Positive tuberculin reactions are not more frequent in underweight children than in others, and latent tuberculosis is evidently seldom, if ever, the cause of underweight in children with malnutrition.

In Table I the dividing line between the groups is the calculated normal weight. Table II contrasts the percentage of positive tuberculins in the total number of children of each age with the percentage of positive tuberculin reactions in children of the same age who were 10 per cent or more underweight.

TABLE II.

Age.	Children tested with tuberculin.		Children one-tenth underweight.		
	Total number.	Percentage positive to tuberculin.	Number.	Per cent.	Percentage positive to tuberculin.
5	73	35.5	1	1.4	0
6	120	45.8	10	8.3	40.0
7	145	53.1	6	4.1	66.7
8	221	68.3	27	12.2	70.4
9	185	67.0	30	16.2	73.3
10	229	72.9	26	11.3	69.2
11	214	75.7	26	12.1	76.9
12	225	83.5	37	16.4	83.8
13	237	83.1	25	10.5	86.0
14	172	89.5	20	11.6	90.0
15	103	93.2	12	11.6	83.3

The percentage of positive tuberculin reactions at each age is practically the same in children with malnutrition and in the general child population. There is no evidence that latent tuberculosis is a factor in the production of malnutrition.

The proportion of children 10 per cent underweight in the younger years, namely, at the ages of five, six and seven, is considerably less than that found in older children, ranging from eight to fifteen years. The factors producing malnutrition make their influence felt by the age of eight, and after this few, if any, new instances of faulty nutrition appear.

Sensitivity to tuberculin makes its appearance during the same period in the child's life as malnutrition, and I am tempted to connect the two as cause and effect. The incidence of malnutrition (10 per cent underweight), however, remains constant at about 12 per cent from the age of eight years onward, while sensitivity to tuberculin continues to increase. Latent tuberculous lesions demonstrated roentgenologically increase between the ages of eight and fifteen without a corresponding increase in malnutrition.

In most instances a positive tuberculin reaction is the only indication of the existence of latent tuberculosis. Not infrequently, tuberculous pulmonary nodules can be demonstrated by roentgenological examination, but they have little significance. Tracheobronchial lesions are recognizable only when the infection has been intense and prolonged.

During the past year, 411 pupils in two schools have had roentgenological examination of the chest. Of these, 307 children from one school were given roentgenological examination without selection. The remaining 104, from the other school, were selected for examination because they gave decidedly positive tuberculin reactions or were underweight. In consequence the group contains a somewhat higher proportion of underweight children than the general population. A diagnosis of latent tuberculosis was made only when the lesion was sharply defined. Since satisfactory *x*-ray films are more difficult to obtain with obese subjects, failure to demonstrate existing lesions is probably more frequent in overweight children.

Table III shows the relationship between the weight and lesions of latent tuberculosis shown by roentgenological examination. The children are divided as in Table I into (*a*) normal or overweight, and (*b*) underweight.

TABLE III.

	Nutrition	
	Normal or underweight.	Underweight.
Total number	219	192
Negative:		
Number	169	140
Per cent	77.1	72.9
Pulmonary nodule:		
Number	32	29
Per cent	14.6	15.1
Tracheobronchial tuberculosis:		
Number	18	23
Per cent	8.2	12.0

Demonstrable tracheobronchial tuberculosis is more frequent in in underweight children (12 per cent) than in others (8.2 per cent). Tracheobronchial tuberculosis may produce underweight in some instances, or underweight children are perhaps less resistant than others to disease in general.

Table IV shows the relationship between the incidence of tracheobronchial lesions and the degree of underweight. The same children are divided into groups based upon the state of nutrition, which is recorded as a percentage of the normal for children of a given height and age.

TABLE IV.—RESULT OF ROENTGENOLOGICAL EXAMINATION.

Percentage of the normal weight for a given height and age.	Total number in group.	Negative.		Pulmonary nodule.		Tracheobronchial tuberculosis.	
		Number.	Per cent.	Number.	Per cent.	Number.	Per cent.
120 or more	40	34	85.0	5	12.5	1	2.5
100 to 120	179	135	75.4	27	15.1	17	9.5
95 to 99	80	54	67.5	14	17.5	12	15.0
90 to 94	71	51	71.8	11	15.5	9	12.7
89 or less	41	35	85.4	4	9.8	2	4.8

If tracheobronchial tuberculosis were a cause of underweight the proportion of children with lesions would increase with increasing degrees of underweight. Children of the group in which the highest percentage of tracheobronchial lesions is found are less than 5 per cent below the calculated normal. A low percengage of discoverable lesions occurred in children who were more than 10 per cent underweight. Children 5 per cent or less underweight are not suffering from malnutrition in the generally accepted sense of the word, and if this group be considered to have normal nutrition, no relationship between tracheobronchial tuberculosis and malnutrition is found. Of the 41 instances of tracheobronchial tuberculosis, 11, or 26.8 per cent, were found in children more than 5 per cent underweight; while of the total 411 children examined, 112, or 27.2 per cent, were more than 5 per cent underweight.

Table V is a list of the 41 children with tracheobronchial tuberculosis. Of these 18, or 43.9 per cent, are well nourished; 15, or 36.6 per cent, are underweight 7 per cent or less; and 8, or 19.5 per cent, are more than 7 per cent underweight.

TABLE V.—CHILDREN WITH TRACHEOBRONCHIAL TUBERCULOSIS.

Name.	Nutrition, per cent.	Name.	Nutrition, per cent.
1. G. C.	137	22. B. G.	97
2. S. P.	114	23. C. L.	97
3. P. A.	113	24. B. A.	96
4. G. B.	110	25. V. W.	96
5. S. L.	109	26. M. M.	95
6. I. L.	108	27. D. G.	95
7. A. S.	108	28. J. S.	95
8. H. S.	107	29. S. R.	95
9. I. F.	107	30. S. C.	95
10. M. S.	106	31. F. F.	94
11. A. F.	106	32. E. G.	94
12. E. B.	106	33. A. B.	94
13. S. P.	105	34. A. D.	92
14. S. S.	103	35. J. C.	92
15. J. F.	101	36. M. B.	92
16. N. M.	100	37. J. O.	91
17. M. G.	100	38. T. E.	90
18. F. L.	100	39. T. Y.	90
19. M. L.	99	40. L. J.	81
20. E. F.	98	41. V. S.	77
21. J. B.	97		

As long as the diagnosis of tracheobronchial tuberculosis rests upon malnutrition, with its attendant symptoms and signs, its occurrence will be frequently undiscovered, and as long as the diagnosis of latent tuberculosis is based upon evidence of malnutrition unsubstantiated by tubreulin tests and especially by roentgenological examination, the diagnosis will often be incorrect. Two bad results may follow: (*a*) The child may be sent to an institution where contact with open tuberculosis is established, and (*b*) the true cause or causes of the malnutrition may be overlooked or ignored.

Ten cases of latent apical tuberculosis were found in school children during the past two years. Physical examination of the chest in each instance failed to reveal evidence of abnormality. Table VI shows age, sensitivity to tuberculin, relation of weight to normal, site of infiltration demonstrated roentgenologically and type of disease in each child.

Three children with latent apical tuberculosis were well nourished, 3 underweight less than 5 per cent, and the remaining 4 underweight more than 5 per cent. Inferences from so small a number of subjects are of doubtful value, but it is probable that latent apical lesions may remain so inactive that they do not produce loss of weight or other symptoms. When they progress all the symptoms

of clinical tuberculosis may appear, as has already happened in one of the cases cited above.

TABLE VI.—CHILDREN OF SCHOOL AGE WITH LATENT APICAL TUBERCULOSIS.

Name.	Age.	Tuberculin reaction.	Nutrition, per cent.	Site of infiltration.
C. F.	13	++ to 0.01 mg. O. T.	106	Left apex
J. W.	9	+ to 0.1 "	103	Right "
M. Sh.	11	++ to 0.01 "	103	Left "
R. Sc.	13	+ to 0.01 "	98	Left "
R. O.	14	++ to 0.1 "	98	Left "
M. B.*	12	++ to 0.01 "	97	Both apices
A. A.	12	+ to 0.01 "	94	Right apex
A. T.	13	+ to 0.01 "	88	Right "
R. Sm.	11	+ to 0.01 "	88	Right "
P. S.	15	+ to 0.01 "	84	Right "

* This child died of pulmonary tuberculosis sixteen months later.

CONCLUSIONS. Weight below normal is not found more frequently in children in whom latent tuberculous infection is shown by sensitivity to tuberculin than in those who give no skin reaction.

There is no evidence of underweight in children with latent tuberculous nodules of the lungs demonstrated by roentgenograms.

In children from 1 to 5 per cent underweight latent tuberculosis of tracheobronchial lymph nodes is more common than in other children. Tracheobronchial tuberculosis is seldom associated with greater loss of weight and is probably not its cause.

A small group of cases indicates that latent apical tuberculosis of adolescence in some instances causes moderate loss of weight but the lesion may occur with no loss of weight.

Underweight has little if any value in the diagnosis of latent tuberculous infection.

CALCIUM STUDIES IN JAUNDICE

With Special Reference to the Effect of Parathyroid Extract on the Distribution of Calcium*

By A. CANTAROW, M.D., S. M. DODEK, M.D.

AND

BURGESS GORDON, M.D.

(From the Medical Service of Dr. Thomas McCrae and the Department for Diseases of the Chest, Jefferson Hospital. A part of the expense of this investigation was defrayed by a grant from Mr. L. A. Swayze, of Canton, Pa.)

In recent years considerable progress has been made in the study of jaundice but comparatively little is known about the details of the chemical processes involved. The tendency to prolonged bleeding in jaundiced patients has long been known. Obviously this fact is of practical importance, since it adds greatly to the operative risk in a condition that often requires surgical treatment. It is generally believed that the hemorrhagic tendency is intimately related to some fault in the availability and utilization of calcium, although the exact mechanism is far from being well understood.

Review of Previous Observations. Calcium exists in the blood stream in several forms: ionized and unionized, diffusible and nondiffusible, free and combined. According to Howland,[1] calcium, phosphoric acid and carbon dioxide are present in the blood in a finely balanced equilibrium, illustrated by the following equation:

$$Ca_3(PO_4)_2 + 2H_2CO_3 \rightleftarrows 2CaHPO_4 + Ca(HCO_3)_2 \leftrightarrows 2Ca^{++} + 2HPO_4^{--} + Ca^{--} + 2HCO_3^{-}$$

Wells[2] states that the calcium salts are held partly in solution, partly in suspension and partly in the form of calcium-ion protein compounds. This condition is extremely unstable; it is subject to alteration by slight changes in any of the several controlling factors. In addition, it is now recognized that the parathyroid hormone plays an important part in the maintenance of the normal calcium level and distribution.

* Read June 1, 1927.

Kirk and King[3] have shown that normally all the common inorganic constituents of serum are diffusible except calcium, is which only from 70 to 75 per cent is diffusible. Grove and Vines[4] state that calcium is present in ionized and combined forms, but that under normal conditions only the ionized form is found in the serum after coagulation, which suggests the importance of combined calcium in the mechanism of coagulation. With such a variety of factors operating to maintain the normal relations of calcium in the blood stream, it is evident that a disturbing element may exercise an effect in several ways. Anything that will alter the calcium level, the hydrogen-ion concentration, the phosphate and the carbon dioxide content, the amount and nature of the proteins, the ratio of diffusible to nondiffusible calcium and of the ionized to the combined forms will cause some disturbance in the functional availability and utilization of calcium. This, however, is not necessarily manifested by any change in the phenomenon of coagulation.

The characteristic feature of jaundice is the presence of an excess of bile pigments and bile salts in the blood. Interference with the normal mechanism of coagulation is perhaps due to the action of these substances, although in advanced cases hepatic malfunction may play a part through the associated diminution in the formation of fibrinogen. Prolongation of the coagulation time does not occur in all patients with jaundice. Lee and Vincent[5] state that it is present only to a slight extent in catarrhal jaundice. These authors have observed no change in coagulation time until the jaundice was of five weeks' duration, and they conclude that such change depends on the intensity of the condition and the amount of available tissue calcium. However, as is well known, the hemorrhagic tendency in jaundice is not incompatible with a normal clotting and bleeding time.

Haessler and Stebbins[6] believe that in some way the bile salts retard the conversion of fibrinogen to fibrin. This factor possibly plays a minor part, however, and for our purposes may be disregarded. Interest is particularly focused on the alteration in the distribution and functional availability of calcium caused by the presence of abnormal amounts of bile pigments in the blood and tissues.

The exact manner in which the pigments operate to diminish the available circulating calcium is unsettled. King and Stewart[7] report that the blood calcium is increased in jaundice owing to obstruction.

This observation was confirmed by King, Bigelow and Pearce.[8] They believe that calcium combines with the pigments as a protective measure in an attempt to counteract their toxicity. This combination appears to fix the calcium in some way and to render it functionally unavailable, probably by lessening the degree of diffusibility and ionization. Kirk and King[3] have found that whereas normally 72.3 per cent of the blood calcium is diffusible, in jaundice this fraction is reduced to 55.2 per cent. They state that the percentage of diffusible calcium in jaundice is subnormal, generally in proportion to the degree of jaundice. They add that this is not the only factor that increases the coagulation time, as similar changes in diffusibility occur in other conditions without affecting coagulation.

It is not unlikely that the nature and amount of blood proteins are of importance in this connection. In certain forms of hemolytic jaundice and in some cases of pernicious anemia the bilirubin content of the blood may be far above the normal renal threshold concentration without appearing in the urine. Bowler and Walters[9] believe that the pigment in such cases is fixed to the plasma proteins to some extent, lessening the diffusibility of the pigment molecule and preventing its excretion. The protein-pigment combination, uniting with calcium, increases the ratio of protein bound to free calcium, altering the normal diffusibility and ionic relations.

When pigment deposition occurs, a considerable amount of calcium which is normally circulating and utilized is fixed in the tissues. Early in the condition this fixation is counterbalanced by the normal calcium reserves but as the intensity progresses and the degree of fixation increases these reserves become relatively depleted and, in a functional sense, an actual calcium deficiency occurs. This accounts for the fact, observed by Lee and Vincent,[5] that early in jaundice there is no change in coagulation time. As they state, such alteration is largely dependent on the amount of calcium in the tissues that can be taken into the blood.

In an attempt to supply this deficiency in jaundice, calcium therapy has been employed with variable results. The success of oral administration is dependent on so many factors that uniformly good results cannot be expected. Lee and Vincent[5] report that large doses of calcium lactate must be given over a period of several days before any alteration in clotting time occurs. Bowler and Walters[10] use small amounts of calcium chloride intravenously for a short

period before operation with apparent success. They report the interesting observation that twice the amount of calcium chloride injected intravenously is required to raise the blood-serum calcium in jaundice to the same level as in normal persons. This, as they say, suggests a calcium deficiency that is not otherwise apparent.

Parathyroid extracts have been proved to exert a profound influence on calcium metabolism. Their administration results in an increase in the amount of available circulating calcium and also perhaps aids in its utilization. In a previous study[11] we observed the changes in the calcium content of the whole blood and of serum at three-hour intervals following the injection of parathyroid extract. The results in six normal cases are shown in Table I. It was noted that the rise in serum calcium generally preceded that in whole blood. Furthermore, it was observed that the curve of whole blood calcium differs materially from that of serum but corresponds more exactly than the latter to the curve of changes in the coagulation time. From this we concluded that the whole blood or plasma calcium content was a more accurate index of changes in the blood following the administration of parathyroid hormone than the serum calcium.

TABLE I.—COINCIDENT DETERMINATIONS OF THE WHOLE BLOOD AND SERUM CALCIUM IN NONJAUNDICED PATIENTS AT THREE-HOUR INTERVALS FOLLOWING THE ADMINISTRATION OF PARATHYROID EXTRACT

	Control.		3 hours.		6 hours.		9 hours.		12 hours.	
Case.	Blood.*	Serum.*	Blood.	Serum.	Blood.	Serum.	Blood.	Serum.	Blood.	Serum.
1	7.64	10.40	7.70	15.70	9.80	11.40	10.05	11.40	12.24	10.90
2	6.43	10.45	11.36	10.22	10.00	9.04	10.28	10.40	8.62	10.24
3	6.60	10.20	6.20	11.30	8.10	11.30	8.10	10.80	11.10	9.60
4	6.52	10.43	5.70	14.54	9.52	11.40	11.20	11.04	10.34	10.76
5	9.54	10.45	11.99	10.00	12.20	9.50	9.80	10.78	8.07	9.13
6	9.61	10.09	10.09	10.09	10.52	8.80	11.17	9.52	8.18	9.04

* Calcium values are expressed in milligrams per 100 cc.

METHODS. The method used for the determination of whole-blood calcium[12] was based on the Clark-Collip modification of the Kramer-Tisdall method for determining serum calcium.[13] The figures are undoubtedly higher than the actual calcium content, owing to the reaction of certain constituents of the stroma of the corpuscles. However, the method was used to obtain a curve showing the changes in blood calcium after the injection of parathyroid extract rather than to determine the absolute calcium values. It

was found satisfactory for this purpose, since the error was a constant one. This was proved subsequently by simultaneous determinations of plasma calcium, coagulation being retarded by the use of heparin.[14] The curve showing the changes in the plasma calcium was found almost to parallel that of the whole blood.

TABLE II.—COINCIDENT DETERMINATIONS OF THE WHOLE BLOOD AND SERUM CALCIUM IN JAUNDICE AT THREE-HOUR INTERVALS FOLLOWING THE ADMINISTRATION OF PARATHYROID EXTRACT

			Control.		3 hours.		6 hours.		9 hours.		12 hours.	
Case.	Type.	Units.	Blood.*	Serum.*	Blood.	Serum.	Blood.	Serum.	Blood.	Serum.	Blood.	Serum.
1.	Hemolytic†	2	10.45	10.45	12.72	11.36	11.17	9.6	9.16	9.31	10.26	8.70
2.	Obstructive	16	10.59	10.00	9.54	9.54	10.18	9.80	10.53	8.43	12.50	9.13
3.	Obstructive	10	12.00	10.02	11.80	10.80	11.20	14.10	11.60	12.00		
4.	Obstructive	3	11.00	9.52	10.50	7.60	13.00	10.00	12.00	10.00		
5.	Obstructive	15	4.80	10.00	7.90	10.00	10.90	10.90	11.60	10.90		
6.	Obstructive	20	7.50	9.30	8.50	9.70	8.80	9.70	9.70	9.70	8.50	9.70
7.	Obstructive	8	8.10	9.55	11.36	9.62	11.47	11.00	14.75	10.40		
8.	Obstructive	7	8.20	11.30	10.20	12.10	11.50	12.40	10.00	11.60		
9.	Obstructive	10	4.90	10.80	9.10	13.10	11.30	12.20	8.40	10.00		
10.	Obstructive	15	8.20	9.30	.	10.00		10.00		9.70		
11.	Obstructive	10	10.96	10.32								
12.	Toxic hepatitis‡	8	4.90	10.50	12.00	11.00	12.40	11.00	11.70	9.00		
13.	Toxic hepatitis	14	7.60	12.00	8.30	13.00	8.30	13.50	10.00	13.50	10.50	14.10
14.	Toxic hepatitis	4	8.20	9.50	...	9.50	. .	10.50	...	10.40		

* Calcium values are expressed in milligrams per 100 cc.
† Amount of bilirubin expressed in units by the van den Bergh method.
‡ Caused by arsphenamine.

TABLE III.—COINCIDENT DETERMINATIONS OF THE WHOLE BLOOD, PLASMA AND SERUM CALCIUM IN JAUNDICE AT THREE-HOUR INTERVALS FOLLOWING THE ADMINISTRATION OF PARATHYROID EXTRACT

	Control.			3 hours.			6 hours.			9 hours.			12 hours.		
Case.	Blood.*	Plasma.*	Serum.*	Blood.	Plasma.	Serum.	Blood.	Plasma.	Serum.	Blood.	Plasma.	Serum.	Blood.	Plasma.	Serum.
8	8.20	14.10	11.20	10.2	17.6	12.1	11.5	18.7	12.4	10.0	17.1	11.6			
9	4.90	8.80	10.80	9.1	17.6	13.1	11.3	20.1	12.2	8.4	16.1	10.0			
10	8.20	9.60	9.30	..	12.0	10.0	..	12.0	10.0	..	11.9	9.7			
11	10.96	18.01	10.32												
13	7.60	17.80	12.00	8.3	18.2	13.0	8.3	19.0	13.5	10.0	19.5	13.5	10.5	20.3	14.1
14	8.20	12.50	9.50	..	15.0	9.5	..	..	..	..	15.2	10.4			

* Calcium values are expressed in milligrams per 100 cc.

The present investigation was undertaken to compare the response to parathyroid extract[15] in jaundiced and in normal persons. The series consists of 14 cases of jaundice; 1 was hemolytic in type, 3

were cases of hepatitis due to arsphenamine and 10 were of an obstructive nature, resulting from either gall stones or carcinoma of the head of the pancreas. The intensity of the jaundice varied from 2 to 20 units of bilirubin as determined by the van den Bergh test. The following determinations were made: clotting time by the Boggs method, and the calcium content of whole blood,[12] serum[13] and, in some instances, plasma. After one control observation the determinations were repeated at three-hour intervals following the intramuscular injection of parathyroid extract. The figures for calcium are presented in Tables II and III.

Results of Authors' Investigations. *Coagulation Time.* The clotting time in all cases before administration of parathyroid hormone was well within normal limits. It ranged between three and five minutes, with apparently no relation to the depth, duration or type of jaundice. The response to the administration of the hormone was similar to that previously observed in persons who were not jaundiced.[11] The clotting time was reduced in all instances, the degree of reduction varying between 5 and 65 per cent. The maximum effect occurred from six to twelve hours after the injection. The rapidity of coagulation did not bear as close a relationship to the whole-blood calcium as it did in the normal cases but it still appeared to be much more dependent on this factor than on the serum calcium level.

Serum Calcium. The normal range of serum calcium is from 9 to 11 mg. per 100 cc. The control values in the normal cases were from 10.09 to 10.45 mg. After the administration of parathyroid extract there was a tendency toward a rise in serum calcium within the first three hours, followed by a gradual return to normal (Table I). The maximum variation occurred three hours after the injection.

In the series of patients with jaundice the control values varied slightly beyond normal limits, from 9.3 to 12 mg. per 100 cc. The response to parathyroid hormone was inconstant: in some instances no rise occurred; in others there were slight rises at varying intervals. We do not attach significance to the absence of an increase in calcium in several cases, since the rise may have occurred in an interval between our determinations. However, the results are interesting when compared with those of normal persons, as illustrated in Chart I. It is seen that the limits of variation in serum calcium are slightly affected by parathyroid extract in jaundiced persons as compared with normal persons.

Whole Blood. The whole-blood calcium in the normal group varied between 6.43 and 9.61 mg. per 100 cc.[12] After the adminis-

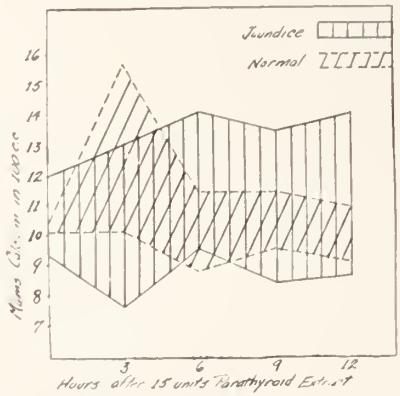

CHART I.—Comparison between the variation in serum calcium in jaundiced and in normal persons before and at three-hour intervals following the administration of parathyroid extract. The area occupied by diagonal lines represents variation between minimum and maximum serum calcium values in normal persons; the area occupied by perpendicular lines represents variation between minimum and maximum serum-calcium values in jaundice.

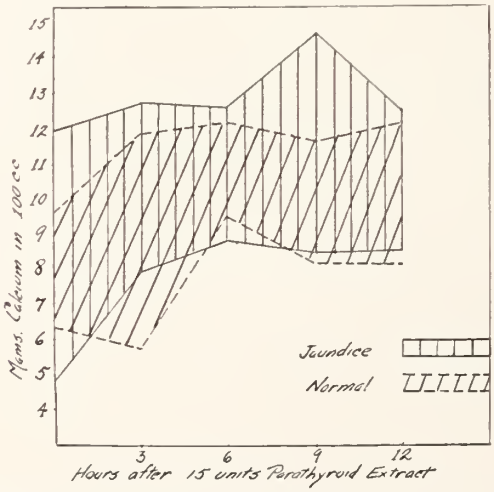

CHART II.—Comparison between the variation in whole-blood calcium in jaundiced and in normal persons before and at three-hour intervals following the administration of parathyroid extract. The area occupied by diagonal lines represents variation between minimum and maximum whole-blood calcium values in normal persons; the area occupied by perpendicular lines represents variation between minimum and maximum whole-blood calcium values in jaundice.

tration of parathyroid extract there was a gradual tendency toward an increase in the blood calcium level, the degree of variation remaining approximately the same as previously. In the jaundiced patients the control values for blood calcium ranged betwen 4.8 and 12 mg. per 100 cc. This wide variation was the first striking difference observed between jaundiced and normal blood. The calcium level bore no relation to the coagulation time, degree, type or duration of the jaundice. The injection of parathyroid extract was followed by a marked decrease in the degree of variation; at the

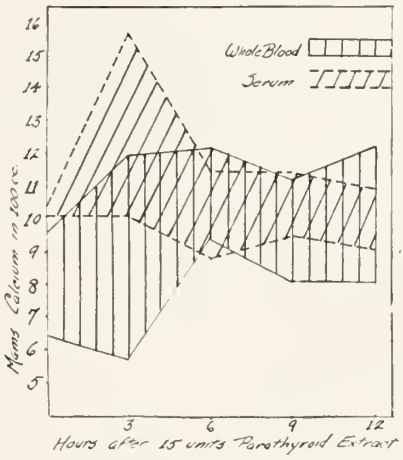

CHART III.—Comparison between the variation in serum and whole blood calcium in normal persons before and at three-hour intervals following the administration of parathyroid extract. The area occupied by diagonal lines represents variation between minimum and maximum normal serum calcium values; the area occupied by perpendicular lines represents variation between minimum and maximum normal whole-blood calcium values.

end of six hours, and particularly after twelve hours, the range in jaundiced persons was practically identical with that in normal persons (Chart II). The relationship between whole blood and serum variations in normal and in jaundiced persons is illustrated in Charts III and IV.

Plasma Calcium. In some cases the calcium content of the plasma was determined. It varied between 8.8 and 18.01 mg. per 100 cc. in the group of patients who had jaundice. The determinations after parathyroid administration were almost exactly parallel to those of whole blood.

COMMENT. In reviewing these data several interesting facts might be noted. However, there are two points which appear to be of particular significance: first, the wide variation in the calcium content of whole blood in jaundice as compared with that of normal blood; second, the fact that after the administration of parathyroid extract this variation is altered, so that at the end of twelve hours the calcium content of the blood corresponds almost exactly to that in normal persons treated in the same manner (Chart II).

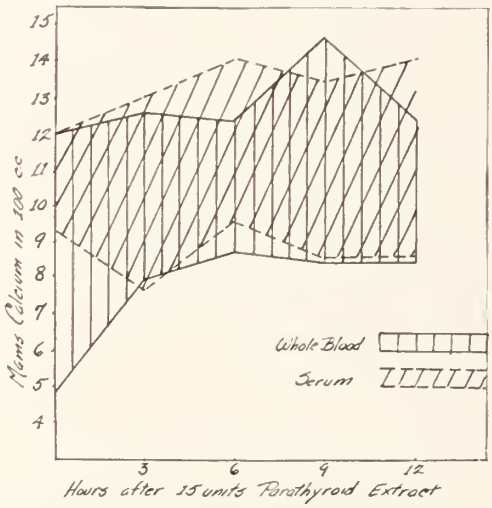

CHART IV.—Comparison between the variation in serum and whole-blood calcium in jaundice before and at three-hour intervals following the administration of parathyroid extract. The area occupied by diagonal lines represents variation between minimum and maximum serum calcium values in jaundice; the area occupied by perpendicular lines represents variation fetween minimum and maximum whole-blood calcium values in jaundice.

The variation in the calcium content of whole blood is undoubtedly of considerable significance. We have stated that the amount seems to be independent of the type, intensity and duration of the jaundice, and that it bears no relation to the coagulation time, but of greater interest perhaps is the fact that it is equally independent of the serum calcium content (Table II). For example in Case 2 the patient had a whole blood calcium of 10.59 mg. and in Case 5, 4.8 mg. The serum calcium content in both cases was 10 mg. per 100 cc. It seems obvious that the amount of calcium which participated actively in the process of coagulation, and which perhaps was incor-

porated in the clot in protein or lipoidol combination, differed greatly in the two instances, being comparatively large in Case 2 and slight in Case 5. This relative fixation of serum calcium irrespective of the blood-calcium level is in all likelihood due to the presence of the bile pigments, which bind the calcium, rendering it functionally unavailable. Since most of the pigment remains in the serum after coagulation, the bound calcium likewise remains. This tends to maintain the relative constancy of the serum-calcium values as compared with those of whole blood in jaundice. The wide variation in the blood calcium must be dependent largely on corresponding variations in the general calcium metabolism of the tissues, in the functionally available and diffusible fractions and in the amount fixed in the tissues by the pigments and excreted with them in the urine. The supposition of functional deficiency is supported by the work of Bowler and Walters[10] referred to previously, in which they observed that much more calcium chloride, given intravenously, is necessary to raise the serum calcium in jaundice to the same level as in normal persons.

The effect of parathyroid extract on the distribution of blood calcium in jaundice is striking. Collip states[16] that in the normal animal, the function of the parathyroid hormone "appears to be that of a regulator of calcium metabolism, and its action is primarily as a calcium mobilizer." We have shown that in jaundice there is a disturbance of calcium metabolism of varying degree, evidenced by the abnormal distribution of calcium in the blood stream. In twelve hours after the administration of parathyroid extract the distribution of calcium is temporarily, practically identical in jaundiced and in normal persons. Evidently the hormone tends to restore the normal balance between blood and tissues in an attempt to remedy the existing functional deficiency. One evidence of the success of this attempt is the reduction of the coagulation time, suggesting an increase in the availability and utilization of the calcium of the blood. Through increasing the functionally available calcium the tendency to slow, protracted hemorrhage of a diffuse nature may be lessened, as noted in incised tissues in jaundice often associated with a normal coagulation and bleeding time. As previously suggested, this favorable effect is perhaps due[17] to a decrease in the permeability of the capillary walls, aided, undoubtedly, by the increased coagulability of the blood following administration of parathyroid extract.

Conclusions. 1. In jaundice it is evident that a functional deficiency in calcium exists, probably due to the increased amount of bile pigments in the blood and tissues.

2. The calcium content of whole blood, plasma and serum and the coagulation time have been determined at three-hour intervals after the injection of parathyroid extract. The observations in jaundice are compared with those in nonjaundiced persons.

3. The serum calcium values varied slightly, ranging from 9.3 to 12 mg. per 100 cc. in jaundice. There was an extremely wide variation, however, in the calcium content of whole blood, the figures ranging between 4.8 and 12 mg. per 100 cc.

4. Twelve hours after the administration of the parathyroid hormone the variation in the whole-blood calcium of jaundiced and nonjaundiced patients was practically identical.

5. Parathyroid extract acts as a mobilizer of calcium; in jaundice it tends to restore the normal distribution and functional availability of this element.

6. The favorable effect of the hormone on the tendency of jaundiced tissue to bleed is due largely to the increased coagulability of the blood and probably to the diminished permeability of the capillary walls, the result of an increase in functioning calcium.

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14. Plasma determinations were made by the Clark-Collip method, powdered heparin being used to prevent coagulation in the specimen of blood.

15. The preparation used was para-thor-mone, supplied by Eli Lilly and Company through the kindness of Dr. G. H. A. Clowes and Dr. J. H. Warvel.

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DISCUSSION

Dr. Hobart A. Hare: As I understand it, you suggest that parathyroid hormones should be given intramuscularly in cases of delayed coagulation time, with the object of shortening coagulation time. The quantity used was 15 units?

Dr. Gordon: The dosage was 15 units, six hours before operation.

Dr. Hare: You have tried this in certain cases of pulmonary hemorrhage?

Dr. Gordon (closing): Yes, sir. In about 58 cases. The failures have been due apparently to the erosion of large bloodvessels or to certain proteolytic changes in the tissues.

THE TREATMENT OF PERNICIOUS ANEMIA WITH LIVER OR AN EFFECTIVE FRACTION OF LIVER.*

The Mary Scott Newbold Lecture
Lecture XVIII

By GEORGE R. MINOT, M.D.
BOSTON, MASS.

In the *Medical Times and Gazette* for the year 1874 (page 581) there appears an editorial entitled "Pernicious Anemia; a New Disease." Since then the disease first described by Addison in 1849, and classically pictured by him in 1856, under the title of "Idiopathic Anemia," has been known as pernicious anemia or Addison's anemia. The term pernicious anemia has occasionally been applied incorrectly, especially in Europe, to cases of severe anemia of various sorts, often due to a known cause. This may be partially explained, because when Biermer, in 1872, first employed the name "progressive pernicious anemia" he applied it to cases of more than one type. Pernicious anemia, heretofore, has been recognized as an idiopathic disease that progressed slowly with remissions to a fatal outcome, usually in the course of a few years.

This communication outlines Dr. William P. Murphy's and my observations concerning the effect of a diet rich in mammalian liver[1,2,3,4] and an effective fraction of liver, either of which can favorably alter the prognosis for essentially all patients with pernicious anemia. The effective fraction of liver has been isolated by Dr. Edwin J. Cohn and with him and our associates we have reported upon it.[5]

Pernicious anemia is characterized by gastric achlorhydria, macrocytosis of the red blood corpuscles, high color-index and a bone-marrow that during relapse shows an extensive proliferation of primitive cells (megaloblasts). We believe that these cells tend to fail to differentiate toward erythrocytes, so that the anemia is explained by a functional ineffectiveness of the bone-marrow, rather

* Read November 2, 1927.

than, as so often suggested, by a process that is fundamentally hemolytic. However, dysfunction of the hemoglobin pigment metabolism is a feature of the disease and leads to an increase of pigments in the blood plasma, tissues and excreta, but this is probably a phenomenon secondary to a defect in the manufacture of red blood corpuscles. Pernicious anemia is further characterized by the frequent occurrence of partial degeneration of the central nervous system and by recurrent glossitis, conditions which may perhaps be dependent upon the same unknown cause that prevents the proper growth of the red blood cells.

Many forms of therapy have been advocated for pernicious anemia but none has resulted heretofore in permitting with regularity a prompt, rapid increase of the red blood corpuscles to four million or more per cubic millimeter in essentially all cases. Our experience up to August 1927 with 150 cases appears to justify the assertion that this is made possible by the daily ingestion of large amounts (125 to 225 gm.) of mammalian liver or a few grams of a certain effective fraction of it, together with an adequate, well-balanced diet.

One hundred and twenty-five of the 150 patients have been treated regularly for from three months to three and a half years with a diet rich in liver. It is only within the past six months that Dr. Cohn has isolated from beef liver the fraction containing the active principle which has been administered to the other 25 patients for periods varying for from two weeks to three months.

The treatment of other sorts of anemia is not pertinent to the present discussion, although some patients with "secondary" anemia can be benefited by a diet rich in liver. On the contrary, there are patients with conditions, such as aleucocythemic leukemia, which may resemble pernicious anemia, and others with various simple sorts of anemia, who are not benefited by this therapy. Thus, errors concerning the value of a diet rich in liver or an effective fraction may be made if the diagnosis of pernicious anemia is not correctly established. This is emphasized because, if the treatment is properly carried out and a distinct improvement in the health of the patient is not apparent in four weeks, it is highly probable that the diagnosis of pernicious anemia is incorrect.

The health of all the 150 patients with pernicious anemia has been markedly improved. They are all living except 4, but none has died with severe anemia. The causes of death have been injury from accident, cerebral thrombosis and infectious conditions. Not

only has nearly every one of the patients responded promptly to the treatment; but, if they have continued with the therapy satisfactorily and complications have not arisen, their red blood-cell count has remained elevated and in almost all instances above 4.5 million per cubic millimeter. The ultimate effect on patients of their continuing to take liver, or something contained in it, together with an adequate diet, can be determined only in the future; but it has already been ascertained that the benefit derived from such treatment is not limited to a few months.

The symptoms and signs common to pernicious anemia, pellagra and sprue, and the fact that some people who develop pernicious anemia have partaken for years of an abnormal diet, are among the reasons that suggested the idea to us that patients with pernicious anemia could be benefited by food of some sort. The feeding of liver has been shown to profoundly affect various bodily processes. It promotes to an unusual degree the growth in size of animals, may enhance the multiplication of cells, hasten the regeneration not only particularly of hemoglobin but also of red blood cells, in animals with anemia due to blood loss, and affects the course of certain diseases. Thus, liver was chosen for these and other reasons as a food that in large amounts might favorably alter the maturation of the primitive bone-marrow cells in pernicious anemia, or, in accordance with Whipple's[6] conception of the disease, correct a defect in the manufacture of the stroma material of the erythrocytes. Peabody's[7] studies on the pathology of the bone-marrow obtained by puncture of the tibia during life indicate that liver feeding probably does act to promote a proper growth of the primitive cells that crowd the marrow in relapse.

We[8] have shown that after the feeding of large amounts of liver there occurs with extraordinary regularity a prompt, temporary, often marked increase in the peripheral circulation of the young red blood cells (reticulated red blood corpuscles or reticulocytes). This behavior of the reticulocytes also suggests that liver stimulates the growth of the primitive red blood cells. The increase of reticulocytes following the ingestion of a given amount of liver will be less the higher the red blood-cell count. The number of reticulocytes that appear in the blood stream also varies with the amount of potent material fed. Further variations in the reticulocyte reaction may occur dependent upon the precise state of the patient, but only under unusual conditions are such variations of importance. Studies

concerning the fraction of liver suggest that there is a maximum production of reticulocytes when material derived from about 500 gm. of whole liver is fed daily. In the untreated case the young red blood cells usually comprise about 1 per cent of all the red blood corpuscles. The reticulocytes start to rise in from three to seven days after the feeding of an adequate amount of liver, and the peak of their rise occurs a few days later. When the red blood-cell count is about 1.25 million per cubic millimeter and about 200 gm. of prepared liver are taken daily, the reticulocytes usually reach a height of about 23 per cent. When the count is somewhat below one million per cubic millimeter the reticulocytes frequently increase to 40 per cent or more of all the red blood cells. If the count is three million or more per cubic millimeter the reticulocytes may rise temporarily only a few per cent or not increase at all. In the two weeks after these cells have reached their peak they progressively decrease to their normal percentage. Their increase is absent only under unusual and special circumstances if the red blood-cell count is below three million per cubic millimeter. The manner in which liver feeding appears to act furthers the conception that the anemia of pernicious anemia is primarily due to a defect in the formation of red blood corpuscles. It seems illogical to attribute its beneficial effect to stopping a hemolytic process.

The increase of the reticulocytes commences before there is a detectable rise in the red blood-corpuscle count. There has been little difference in the rate at which the red blood cells rose or the level they reached when liver therapy was started during a first relapse or a subsequent one. Data concerning the rate of increase of the corpuscles for all of the 108 of the 125 patients who have been treated with whole liver and an adequate diet, and whose red blood cells were below 2.7 million per cubic millimeter when they were first placed on the diet, are as follows: The average red blood-cell count before the diet was begun was 1.50 million per cubic millimeter. After liver had been taken for about one month, two months, and between four and six months, the average count was respectively 3.36, 4.25, and 4.65 million per cubic millimeter. The average figures for the counts of the patients who took the largest amounts of liver are definitely greater than for all cases including those in patients who took relatively little liver.

The hemoglobin increase often has resulted in causing the color index to become less than normal as the counts rose over four million

per cubic millimeter. The average diameter of the red blood cells has frequently become normal and sometimes smaller than normal.[9] The blood platelets rapidly increased as the young red cells entered the blood stream and the white blood cells were favorably affected. Increases of polynuclear eosinophil cells, sometimes of a marked degree (over 30 per cent), have been observed occasionally some weeks after the patient commenced to take liver. The increases have been greater in patients taking raw than cooked liver and have been more often absent or only slight in individuals taking liver extract. The significance of the eosinophilia is yet to be determined. The excess of bile pigments in the plasma decreased so that often within three weeks after liver therapy was commenced the icterus index became normal.

The red blood cells of 119 of the 125 patients receiving whole liver have reached over four million per cubic millimeter. The corpuscles of the remaining six have increased more than two million and have not significantly decreased. After a high level of the red blood cells has been obtained, temporary, seldom marked, decreases below four million per cubic millimeter have occurred in about 15 per cent of the cases. The decreases were almost always associated with a partial or complete omission of the prescribed amount of liver. Failure to take a sufficient amount of liver, or the presence of a complication such as an infectious process or cirrhosis of the liver, may cause the count to fall, or cause it to rise, less rapidly than might be expected.

The few patients whose counts have not reached four million per cubic millimeter and the few who have responded unusually slowly, either have not taken the diet properly or have had some complication. In 2 it was more than six weeks after the diet was started before the red-cell count increased. The slow response of at least 1 of these patients was probably due to the marrow having been previously injured by multiple transfusions of blood.

At the end of a year the average red blood-cell count for all of the 60 patients who have taken the diet for this length of time was 4.52 million per cubic millimeter. Eighteen patients have taken the diet for as long as two years, and their average count was then 4.65 million per cubic millimeter. Only 3 patients have had the diet for three years or longer. When three years had passed their average count was 4.81 million per cubic millimeter. At any given time after the diet had been taken for three months or longer, the counts

of over a third of all the patients, including those who have taken the diet poorly, have been above five million per cubic millimeter, which further indicates that the high average counts have been persistently maintained.

Symptomatic improvement is usually rapid and striking and becomes evident in a few days, or when the reticulated red blood cells have risen. The appetite has improved rapidly and often becomes ravenous, perhaps partly dependent upon the ability of liver feeding to decrease the blood-sugar.[10] The symptoms of anemia *per se* disappeared, and two months after starting the diet the patients as a rule looked well, except for disorders of the central nervous system. However, muscular weakness not associated with spinal-cord degeneration has occurred in a few patients after the red blood cells were present in normal numbers. The typical tongue symptoms usually vanish quickly and have recurred only in mild form in a few patients who have not taken the diet well. However, achlorhydria has persisted in the 20 cases examined. Symptoms referable to the central nervous system have rarely definitely progressed and have not developed under adequate dietary therapy. Numbness has disappeared, and occasionally bone vibration sense has increased. Coördination of the extremities has improved frequently and sometimes to a marked degree. The greater part of such improvement must be attributed to increase in muscular strength secondary to the continuous high level of the red blood corpuscles. Exercise to retrain and develop muscles will be found distinctly helpful for these patients.

The nature of the material effective in pernicious anemia is being investigated by studying the influence of feeding fractions of liver. The occurrence or absence of a response of the reticulocytes gives information within ten days of the value of the substance administered. Dr. Cohn has progressively subjected liver to a chemical dissection, as a result of which the protein, carbohydrate and lipoidal constituents have been eliminated without loss of the active principle. A fraction less pure than has been produced for experimental purposes can readily be manufactured which contains less than 1.5 per cent of the liver.

Although the investigations which are in progress have not yet resulted in the isolation of the active principle, certain of its characteristics have been determined. It is readily soluble in water, but is precipitated by alcohol or acetone, and is insoluble in ether.

Though free of proteins, the purest fractions thus far produced are richest in nitrogen. They are also free of carbohydrates, lipoids and iron. The indications are that the effectiveness of the extracts is not due to known vitamins and that the active principle is in the nature of a polypeptid or a nitrogenous base.

A few grams of the purest fraction in aqueous solution taken daily by mouth produces a prompt, marked, temporary increase of reticulocytes, causes a rapid rise of the red blood corpuscles, and appears to act with apparent specificity to improve markedly the health of patients with pernicious anemia. This has been the effect in the 25 patients first treated with a potent fraction, and data concerning them are entirely comparable to those for patients treated with whole liver. The patients have taken the fraction in some instances until after their red blood-cell count had reached normal and remained so for several weeks, and in others until the count has increased over two million cells per cubic millimeter. Subsequently, owing to lack of material, they have taken daily whole liver. The individuals have also taken as soon as possible an adequate, well-balanced diet, and this is of importance. There is some evidence at hand to suggest that after the red blood cells have increased about two million per cubic millimeter and the hemoglobin about 30 per cent, the further rises will be more rapid if the diet is rich in vegetables, fruits and red meat (foods that Whipple and Robscheit-Robbins[11] have shown enhance blood regeneration in dogs with anemia from blood loss), than if it contains chiefly concentrated carbohydrate foods and others that do not influence blood regeneration.

The use of a liver fraction simplifies treatment, especially for very sick patients. Both whole liver and the liver fraction have caused very ill patients, including some in a comatose condition, to become rapidly better. However, it must be appreciated that patients may first receive treatment when their disease has progressed so far that death will ensue or fatal complications arise before there is sufficient time (about a week) to adequately influence the bone-marrow by the administration of a suitable amount of liver or a preparation of it. On the contrary, experience indicates that pernicious anemia patients with little anemia, but with symptoms of a lack of the sense of well-being, gastrointestinal or neurological symptoms, can be benefited by the therapy.

It will require often much time, tact and sympathy to persuade the patient to take the correct food. Giving little other food than

mammalian liver (by stomach tube if necessary) has permitted individuals soon to be able to take the full amount of liver (200 gm. cooked or as raw liver pulp) and an adequate diet. The few patients who have responded slowly have taught us the importance of perseverance and the desirability of forcing large amounts of liver for months; 100 gm. a day may produce rapid improvement, but double this amount is more apt to do so. Cooked liver may be served in any way that pleases the patient, but prolonged boiling is to be avoided. As a daily portion the broth from 500 gm. of liver is efficacious. Raw liver served as a finely divided pulp has been found suitable. It is simple to take and can be swallowed rapidly. The daily dose may be divided into two portions and taken mixed with water or fruit juice, mid-morning and mid-afternoon. The possibility of parasitic infection is recognized but seems remote, at least in the temperate zone. Kidneys have been demonstrated to contain the active material and may be substituted for liver.

The problem of how much liver the patient should continue to take after his red blood-cell count becomes normal remains unsolved. Some patients require more than others. One hundred and fifty grams three times a week has apparently been sufficient in some cases to maintain good health, but more is usually desirable. The necessity of prolonged continuation with a diet rich in liver, or a suitable fraction of it, is emphasized. It is possible that if an excess of the effective material in liver is taken constantly for a long time it might cause some exhaustion of the bone-marrow and lead to anemia. The consequences of feeding the extract for prolonged periods of time remain to be investigated. It is probable that after what appears to be a deficiency which leads to pernicious anemia has been supplied by the specific substance in liver, the adequate production of red blood corpuscles containing normal amounts of hemoglobin will necessitate other dietary factors.

The diet need not contain any unusual amount of protein or number of calories, but it is advised that it be rich in fruits and green vegetables and contain red meat. Large quantities of fat may upset digestion and cause undesirable gain in weight and may be wise to avoid. Excess of fat has not inhibited blood regeneration. It is desirable to curtail sweet foods and those rich in starch. They have no favorable effect on blood formation and intensify the colonic indigestion that often complicates the disease. The diet should not be a standardized one for all patients, but should be prescribed for

each individual. It must contain large amounts of liver, kidney or an appropriate equivalent, such as the effective liver extract, and foods to supply liberally, but not excessively, all requirements of the body. Treatment other than dietary and the administration of a preparation of liver is subsidiary.

SUMMARY. One hundred and twenty-five cases of pernicious anemia have been treated with regularity for from three months to three and one-half years with a diet rich in mammalian liver (180± gm. daily), and 25 additional cases have been recently treated with a few grams a day of a potent nonprotein, noncarbohydrate, nonlipoidal fraction of liver which Dr. Cohn has isolated. The results obtained indicate that, if either form of therapy is properly carried out, great improvement in health will follow, usually promptly and rapidly, in essentially all patients with the disease. Kidney feeding is also efficacious.

In almost all of the cases the red blood-cell count has reached above four million per cubic millimeter. The counts have remained at or above this level for as long as adequate treatment has been continued, except for slight temporary decreases due to infections. At the end of a year the average count (60 cases) was 4.52 million, and at the end of two years (18 cases) 4.65 million per cubic millimeter. The potent fraction of liver acts with apparent specificity in pernicious anemia, and in a similar manner as does whole liver. The use of the fraction simplifies treatment. Even so, all patients must take an adequate, well-balanced diet.

NOTE.—Since this paper was prepared in October, 1927, more information concerning the subject has been obtained. More complete and recent information appears in two articles in the May, 1928, issue of the *American Journal of the Medical Sciences*, and in a paper in the May, 1928, issue of the *Journal of Biological Chemistry*.

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THE PATHOGENESIS OF GASTRIC AND DUODENAL ULCERS*

The Mütter Lecture

By E. STARR JUDD, M.D.
ROCHESTER, MINN.

At times one is inclined to become discouraged over the apparent lack of progress made in our knowledge of disease. However, if we look back just a few years to the time when Doctor Mütter was Professor of Surgery at Jefferson Medical College (1841–1856) we realize that something has been accomplished. Several years after Doctor Mütter's time (1885), Pepper's "System of Medicine" appeared containing a chapter on gastric ulcer written by Dr. William H. Welch, which remains to this day the best résumé of the etiology and pathogenesis of this condition in the literature. Duodenal ulcer was not even considered in those days.

I chose the subject "The pathogenesis of gastric and duodenal ulcers" for the lecture which you have so kindly invited me to deliver, hoping that, although I had little information to give, I might clarify my own opinions and possibly be of help to some one else.

Duodenal and gastric ulcers are separate and distinct lesions. The cause or causes of these ulcers are not as yet known. In order to treat the lesions in a rational manner an attempt must be made to correlate and clarify the many conflicting views regarding their nature and origin.

The problem of the origin of gastric and duodenal ulcers has been studied from the clinical, experimental, anatomic and pathologic points of view. The approach to such study is difficult, especially experimentally, because these lesions are peculiar to man. Cohnheim divided the problem into two parts, namely, the cause of the original defect and the cause of chronicity. This division of the problem simplifies it in some ways. That an acute ulcer may become chronic is a reasonable assumption. Nevertheless it is an open question

* Read December 7, 1927.

whether the occurrence of chronicity involves fundamental principles other than those involved in the origin of the original defect.

During the first decade of this century the question of the origin of ulcers centered around the theories of Rokitansky and Virchow, who believed that ulcers are the issue of the combined action of localized vascular disturbance with focal nutritional changes, and the digestive action of the gastric secretion on the devitalized tissue. In principle, this hypothesis is held today.

Aschoff stresses the Cohnheim theory and attempts to explain the cause of erosions on the one hand and the factors producing chronicity on the other. He differentiates two types of erosions, those of the fundus of the stomach and those occurring along the lesser curvature in the gastric pathway. Only the latter have a direct bearing on this problem. Erosions in this area may be due to spastic conditions of the channel itself or to arterial blocking of a spastic, embolic, or arteriosclerotic nature. The factors tending to produce chronicity are grouped together under the mechanical-functional theory of Aschoff.

The structure and function of the gastric pathway must first be considered. It is formed usually of four longitudinal folds extending along the lesser curvature of the stomach from near the cardia to the pyloric canal, where it becomes obliterated rapidly. The muscle coats are so arranged that contraction causes the structure to assume the form of a channel through which at least a portion of the contents of the fundus may be drained into the pyloric canal. This channel represents, according to Bauer, a rudimentary gullet. In this area the mucosa is thinner and more firmly fixed and the blood-supply is poorer, especially at the isthmus. Also less mucus forms in the gastric channel than in other portions of the stomach. These factors help to explain why erosions of the gastric channel have a tendency to become chronic.

The form commonly assumed by gastric ulcer is due to peristaltic contractions which tend to push the mucosa over the edge of the ulcer, producing an overhanging edge and allowing the gastric juice to stagnate and burrow, thus giving the ulcer the characteristic funnel shape and sloping course. The significance of this explanation is perhaps overestimated as many gastric ulcers vary greatly from this so-called "characteristic type." Moreover, the explanation does not hold for duodenal ulcers.

The characteristic lesion of peptic ulcer in human beings is

usually on the lesser curvature of the stomach in the pyloric mucosa or just distal to the pylorus in the duodenal mucosa. It thus occurs in the region of the gastrointestinal tract which may be exposed to an acid medium and on which the gastric contents may impinge with considerable force during the passage of food through the stomach. These two considerations form the basis for Mann's experimental investigation of peptic ulcer.

TABULATION

Experimental Production of Peptic Ulcer (McCann)

- Vascular
  - Emboli
  - Ligation of vessels
  - Vascular spasm
  - Portal insufficiency
- Bacterial
  - Ingestion of bacteria
  - Intravenous injection of bacteria
  - Specific organisms and foci of infection
- Toxic
  - Pharmacologic toxin
    - Systemic
    - Local
  - Biologic toxins
    - Burns
    - Gastrotoxin
    - Antitoxin
    - Bacterial products
  - Miscellaneous
    - Heat
    - Cold
    - Radiation
- Trophic
  - Central nervous system
    - Vagus section
      - Cervical
      - Thoracic
      - Abdominal
    - Stimulation and irritation
  - Sympathetic nervous system
    - Splanchnics
    - Celiac plexus
  - Combined vagus and sympathetic section
- Glandular
  - Adrenal } Single and combined
  - Thyroid } Single and combined
  - Liver
- Digestive juices
  - Chemical
    - Hydrochloric acid
    - Bile and bile salts
  - Surgical
    - Exclusion of bile
    - Exclusion of pancreatic juice
    - Duodenal drainage
      - Ileal
      - Gastric

Previous investigations had shown that it was difficult or impossible constantly to expose the gastric mucosa to an acid medium by adminstering acid. It was believed that such exposure might be brought about by eliminating the secretions poured into the duodenum beyond the pylorus, which tend to neutralize the acid that has not been neutralized in the stomach. Such elimination has been accomplished by various types of experiments, such as removal of the duodenum, transplantation of the common bile-duct and pancreatic ducts, and a combination of these two procedures. A varying percentage of ulcers was produced in the duodenum or jejunum by each of these methods, depending on the type of operation. How-

ever, the most satisfactory method for producing subacute or chronic peptic ulcer experimentally is the one devised by Mann, that is, to sever the pylorus, invert the distal end of it, sever the jejunum a few centimeters distal to the ligament of Treitz, anastomose the distal end to the pylorus, and anastomose the proximal end of the jejunum to the ileum 30 to 60 cm. from the termination of the ileum. These procedures cause the gastric contents to be expelled from the stomach into the jejunum without becoming mixed with the secretions poured into the duodenum, the bile the pancreatic juice, and the duodenal juice, which are drained into the ileum.

Peptic ulcer develops in almost every animal in which the foregoing procedures have been carried out. The time after operation in which the ulcer develops varies. In some instances the ulcer has perforated as early as ten days after operation, and in others it has not started to develop after three months or longer. The ulcer is almost always in the intestinal mucosa just distal to the suture line, lateral and posterior to the central axis of the organ. Usually only one ulcer is present, although sometimes two and rarely three develop. In the early stage the ulcer never involves the suture line, although retrograde formation may occur which will eventually reach the suture line. The ulcer appears grossly like the peptic ulcer noted in man. It is more or less circular and punched out with a hard base and overhanging edges. Many of the ulcers perforate and if death from peritonitis does not occur, closure will be effected by adjacent coils of the intestines, omentum, pancreas, or gall-bladder. Perforation has occurred into the colon and gall-bladder and through the anterior abdominal wall. Hemorrhage from the ulcer may result fatally. In some instances Mann and his associates have observed the development of these ulcers from the time when only slight injury to the mucosa was noticeable until they became large, indurated and perforated and had existed for from four to six months. Macroscopically the ulcer first appears as a saucer-like depression in the mucosa. In the earliest stage all that can be seen is a small area covered with homogeneous gray material. Gentle sponging uncovers a slight depression where the surface of the mucosa has disappeared and where bleeding is profuse. This gray covering has been noted on all ulcers that have not penetrated beyond the mucosa at the time of examination. Often it is the only means of recognizing "beginning ulcer," the mucosa otherwise appearing normal. After the mucosa has become eroded

the process of ulceration may proceed quickly until the entire wall of the intestine is perforated or there is a hard indurated base. After an ulcer has once started it develops rapidly; the minimal time necessary for penetration through the mucosa is only a matter of hours. An ulcer may develop in mucosa which macroscopically appears normal, and may perforate and produce death from peritonitis within from forty-eight to sixty hours. The typical appearance of a chronic ulcer may be present within three weeks after the onset. An ulcer that has existed for five months may appear the same macroscopically as it did one month after onset.

The earliest ulcers examined microscopically have shown the covering of mucosal cell débris and evidence of considerable hemorrhage between the underlying and adjacent tubules. The ulcer begins at the surface of the mucosa and at first only the mucosa is involved. As the mucosa disappears leukocytic infiltration occurs and the lesion quickly penetrates below the muscularis mucosa. The ulcer then assumes its well-known characteristic appearance.

The ulcers do not heal spontaneously but can readily be made to heal by partial or complete protection from the gastric juice or by draining the contents of the duodenum back over them. Healing was most readily effected by closing the pylorus and draining the stomach by gastroenterostomy, placing the stoma in the jejunum a few centimeters distal to the ulcer. Within four days the base of the ulcer is clear and a thin protective covering of fibrin has formed over it. Within ten days the ulcer has greatly decreased in diameter and depth and the mucosa has begun to grow in from the edges. On the twentieth day three-fourths or more of the base is covered with mucosa. After the thirtieth day it is almost impossible to find the site of the ulcer.

Mann studied healing ulcers both macroscopically and microscopically. Macroscopically the first evidence of healing is the disappearance of exudate and débris from the base, and the formation of a smooth surface. Then the ulcer appears to grow shallow, owing to filling in of the base by new granulation tissue. The overhanging edges of mucosa and the granulation tissue completely fill the base of the ulcer. Gradually the edge of mucosa grows from the periphery toward the center, usually pushing the granulation tissue up and out like a plug. At first the mucosa is thin and smooth. Gradually it thickens and is thrown into folds. Inflammation in the base subsides and the hard induration disappears. The development of the

granulation and connective tissue, and the growth of the mucosa, can be closely observed microscopically. As the base of the ulcer becomes clean a thin protective covering appears. Then the leukocytes gradually disappear and granulation tissue develops. The disappearance of the leukocytes is probably dependent on the protective covering which prevents bacteria from penetrating the base of the ulcer. The granulation tissue is usually extremely vascular and it may readily be surmised that if the base of a healing ulcer is injured, copious hemorrhage might occur. The connective tissue proliferates and usually a thin limiting membrane forms over the base of the ulcer. The mucosa grows in from the edges over the surface of the granulation tissue. At first the mucosa consists of only a single layer of flat or cuboidal cells on a basement membrane. Often the growing edge of mucosa is under an overhanging protecting edge of the plug of granulation tissue in the center. The granulation tissue grows like a toadstool with the pedicle at the center of the ulcer and the top overhanging the edge of mucosa. As the cells of the mucosa increase in number and size, they become typically columnar and the smooth edge is thrown into small folds which carry the newly formed connective tissue with them. At first the folds are small and wavy, but as the mucosa grows, the typical normal villi appear. The growing edge of mucosa is very fragile and it can readily be seen how easily it would be destroyed by the passage of the gastric contents over it. As a matter of fact, Mann says he has never been sure we were studying the ultimate growing edge because, with the most careful technic in preparing the specimen, it always seems as though some of the edge is lost.

Early in the healing of an ulcer the macroscopic changes are more noticeable than the microscopic. Histologically the protected ulcer presents definite variations from the specimen removed at operation but careful study reveals a similarity of the processes in the two specimens. This observation reveals that the healing processes are active in ulcers at all times, but when an ulcer is not protected from the gastric contents the newly formed cells are destroyed before they can become an integral part of the tissue.

An ulcer will also heal if it is only partially protected from the gastric contents. Morton found that healing occurred following gastroenterostomy. Furthermore when the duodenal contents were drained back over the ulcer, healing occurred. However, in both instances the healing process is irregular and in many instances

some of the granulated tissue is injured and hemorrhage into the base of the ulcer may occur. According to Mann the following points are pertinent:

1. Chemical and mechanical factors appear important in regard to the process of development of peptic ulcer.

2. The time necessary for an ulcer to develop and become chronic is relatively short.

3. The ulcer begins at the surface of the mucosa.

4. All complications observed in ulcers of human beings have been noted in the experimental ulcer.

5. The ulcer heals readily when protected from the gastric contents.

6. The healing process is characteristic.

7. If for any reason the granulation tissue in the base of a healing ulcer is injured, profuse hemorrhage may occur.

8. Observations on the process of formation and the process of healing of ulcers disclose the reason for chronicity. They become chronic because the new cells formed in the process of healing are destroyed by the acid or swept away by the passage of the gastric contents before they have had an opportunity to establish themselves as an integral part of the tissue. The following prove the foregoing statement: (*a*) Ulcers heal with remarkable rapidity after being protected from the gastric contents; (*b*) new tissue formed in the healing process is easily injured or destroyed mechanically, as by gentle washing with tap water, and (*c*) reparative processes are at all times active in the ulcer. They probably begin immediately after the injury. The destructive process which produces the ulcer and the attempts at repair go on simultaneously. However, each protective covering formed over the base is destroyed before healing can take place beneath. As the delicate cells of the mucosa develop in the edge of the ulcer they are killed or washed away. It is thus evident that the chemical and mechanical factors are potent in the development of chronicity.

An interesting observation was made by Bollman and Mann in their researches on the liver. In experimental animals duodenal ulcer followed the performance of partial hepatectomy or Eck fistula, and in some instances after ligation of the common duct. Ulcer occurred often enough to interfere considerably with the experiments. In many instances the ulcer perforated and the animal died. It is possible that trauma is a factor in the production of some of

these lesions; in others it probably is not a factor. In the ligation of the common duct the bile is withheld from the duodenum and this may be a factor in the production of ulcer, but in partial hepatectomy and Eck fistula the only change would be in the tissue of the liver or in the circulation to the liver. Whether or not chronic ulcer of the duodenum may be brought about by some disturbance of the function of the liver is an interesting speculation.

Morton, experimenting on dogs, first demonstrated the inherent power of tissue of the normal stomach to heal. He then excised small areas of gastric mucosa in different regions of the stomach and instituted surgical duodenal drainage after the technic of Mann. In 50 per cent of twenty experiments gastric ulcers of subacute and chronic type were produced on the lesser curvature while similar areas on the greater curvature healed. In a series of experiments in which surgical duodenal drainage was instituted and followed two weeks later by the excision of small areas of gastric mucosa, all areas showed delay in healing. In 62.5 per cent of the more prolonged experiments gastric ulcers of subacute and chronic types were produced on the lesser curvature while similar areas on the greater curvature healed. In another series of experiments patches of jejunum with intact circulation were transplanted into the stomach at various points. These remained normal. After varying periods surgical duodenal drainage was instituted. Typical chronic ulcers developed in three of five patches on the lesser curvature. Ulcers did not form in patches in other areas. These observations again emphasize the significance of the chemical and mechanical features in the formation of ulcer.

Von Bergmann enlarged the outlook on the origin of ulcer from a clinical point of view. According to the neurogenic theory of von Bergmann, ulcers are the result of dysfunction of the vegetative nervous system. Nervous factors produce spasm either of the muscle or vessels in localized areas with resulting circumscribed ischemia of the mucosa from which localized mucosal defects arise. Such defects tend to produce further nervous irritability and thus favor chronicity. It is pointed out that certain portions of the stomach, the cardia, the angle, and the pylorus, are more plentifully supplied with nerves than other areas, so that the generally specific site of the lesions is not necessarily an argument against the theory. Much effort has been expended to prove or disprove the neurogenic theory by means of animal experiments. Various investigators

have injected pilocarpine and other spasm-producing drugs, removed the celiac ganglion, cut the splanchnic nerves, and by long-continued stimulation of the vagus have been more or less successful in producing acute or even chronic ulcers in animals. The results, however, are so variable that it is difficult to draw conclusions and even in the most successful experiments it must always be remembered that the conditions met with are not necessarily applicable to man.

Koennecke has shown in dogs that the secretion of the pyloric glands functions in some way to stimulate the acid secretion of the fundus. Clinical observations have shown that pyloric exclusion favors the formation of a particularly powerful gastric juice; the procedure, therefore, is avoided and pyloric resection is advocated by surgeons who lay great stress on the importance of the acid factor in the cause of ulcer.

Konjetzny's theory of the inflammatory origin of ulcer is based on gross and microscopic study of tissue resected at operation. Konjetzny, Orator, Puhl and others observe that in many cases of gastric and duodenal ulcer there is more or less extensive gastritis and duodenitis. According to Konjetzny and Puhl the observation in a single resected specimen of all the stages from simple erosion to chronic ulceration gives the impression that all the steps in the pathogenesis of chronic ulcer are represented. In several patients giving a long history of ulcer only duodenitis and gastritis were found. This discounts the claim that gastritis and duodenitis are necessarily secondary to chronic ulcer. Puhl believes that gastritis and duodenitis are primary to the formation of chronic ulcer. Exogenous irritative factors and constitutional states play an important part in causation. Such lesions are common and usually subside, but in the presence of certain conditions, as the mechanical functional factors of Aschoff, chronic ulcer develops.

Gastric resection is not often performed in cases of duodenal ulcer. In duodenal ulcer, however, a considerable portion of the duodenal cap together with the anterior half of the pyloric sphincter and a small adjoining piece of stomach is frequently excised. In studying the specimens thus obtained one will be surprised at the frequency with which duodenitis appears as the only lesion. In a recent report[7] of 26 cases, in spite of a long history typical of ulcer and with positive roentgenograms, only duodenitis could be demonstrated pathologically. No evidence of ancient or recent chronic ulceration was found in any of the specimens. In gastric ulcer it is

often possible to demonstrate more or less widespread gastritis. That inflammation plays an important part in the ulcer problem is further shown by the fact that in 12 cases operated on for jejunal ulcer true ulcer was not found. In these cases the jejunum was thick, distended, friable, red and edematous, and the mucosa was covered with tiny hemorrhagic areas. Undoubtedly duodenitis is a surgical and pathologic entity, but its relationship to chronic duodenal ulcer is not clear, neither is the full significance of gastritis and jejunitis as entities appreciated.

The pathologic data in cases of duodenitis call attention to the work of Rosenow, as the lesions closely resemble those he produced in animals by injecting specific strains of streptococci. Rosenow has recovered these strains from the tonsils and infected teeth in patients with ulcer of the stomach or duodenum and also from inflammatory duodenal tissue removed at operation. Puhl was able to demonstrate organisms in tissues showing gastritis and duodenitis but could not culture them. Recently Nickel and Hufford recovered a green-producing streptococcus from tissue removed at operation in 10 of 11 patients having ulcer. They say that the localizing power of the streptococcus so constantly found in the foci of patients suffering from ulcer is further illustrated by the results obtained from this series of patients with ulcer. At operation 4 of the 11 patients were found to have gastric ulcer, 4 duodenal ulcer, and 3 gastrojejunal ulcer. In 10 of the 11 cases, the green-producing streptococcus was isolated from the resected ulcers, and all ten strains produced specific lesions in rabbits. Twenty-eight of 31 rabbits injected intravenously with these strains developed hemorrhagic erosions or ulcers in the stomach or duodenum. The foci of these 11 patients were investigated in the same manner as in the first series. A tonsil or nasopharyngeal culture was obtained from every case and nine of the eleven cultures produced positive gastroduodenal lesions in experimental animals. Of the 22 rabbits injected intravenously with these cultures of the tonsils and nasopharynx in thirteen lesions of the stomach or duodenum developed. Two of the cultures obtained from the teeth of these patients produced gastroduodenal lesions in rabbits, and in 3 of the 8 rabbits injected with the cultures of the teeth gastroduodenal lesions developed. Similarly in this series one of six cultures of the prostate harbored streptococci and when a rabbit was injected with this culture gastroduodenal lesions likewise developed. Thus, in 62 animals injected

with culture of ulcers and foci of the patients in this series, lesions of the stomach or duodenum developed in 45 (73 per cent) in contrast to lesions of the gall-bladder, heart and lungs (3 per cent), intestine (5 per cent), and kidneys (7 per cent).

Some of the foregoing theories, although ostensibly referring to both gastric and duodenal ulcers, seem to be more directly applicable to the former. The lesions are distinct. Duodenal ulcer occurs about ten times as frequently as gastric ulcer. No doubt many of the same factors play a part in the etiology of both gastric and duodenal ulcers. Probably some of the factors involved in the production of these two lesions are different.

Ulcers form in the jejunum not only following gastroenterostomy for ulcer of the duodenum but also after resection of the stomach for duodenal ulcer. Furthermore, ulcers recur in the jejunum after resection of the stomach for jejunal ulcer. Recurrence in the jejunum has been known to occur repeatedly, even when several resections have finally resulted in the removal of the greater part of the stomach. These experiences tend to support Mann's experiments and the mechanical and chemical theory of the cause of ulcer, but do not prove that the removal of the lesser curvature will prevent their formation. Experience seems to show that there is a tendency for the formation of ulcers in certain individuals and that no matter what treatment is undertaken recurrence is almost certain to follow. The factor responsible for this has not been determined and its discovery will yield valuable information concerning the etiology and pathogenesis of ulcer.

The final word concerning gastric and duodenal ulcers has not yet been spoken. The multiplicity of hypotheses advanced to explain their origin indicates that many factors play a part, differing perhaps in each case. In the presence of conflicting views regarding the origin of ulcers, different forms of treatment are justified only on the basis of experience. A sharp line is drawn between gastric and duodenal ulcers. The problem of cancer enters into the consideration of gastric ulcer. No matter how small the percentage of gastric ulcers affected by cancer, it adds to the seriousness of the disease. Cancer almost never occurs in the first part of the duodenum. Many duodenal ulcers heal permanently under medical treatment. The element of seriousness in duodenal ulcer is introduced by the occurrence of jejunal ulcer in a small percentage of cases in which gastroenterostomy has been performed. Because of the menace of

jejunal ulcer gastroenterostomy is avoided whenever possible, especially if patients are young and there is a history of hemorrhage. In such cases, it is preferable, whenever possible, to excise the cap of the duodenum and the anterior part of the pyloric sphincter and close the opening as a gastroduodenostomy. Gastric ulcers are either excised and gastroenterostomy performed, or partial resection of the stomach is carried out. Methods of treatment vary with the problem presented by the individual case and conservatism should be the dominating influence except when the question of cancer enters the problem.

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SURGICAL TREATMENT OF PULMONARY TUBERCULOSIS*

By RALPH C. MATSON, M.D.
PORTLAND, OREGON

In spite of the discovery of numerous sera vaccines and chemotherapeutic substances, whose originators claimed, or intimated, that they were cures for pulmonary tuberculosis, rest still remains the most valuable remedy in the treatment of this disease as in the treatment of tuberculosis of the larynx and joints. It is a common observation that as a result of bedrest only many progressive febrile types of pulmonary tuberculosis are converted into retrogressive afebrile forms. Thus, functional rest of the lung as a result of bedrest, or at least body rest, is admittedly the most important factor in the hygienic, dietetic, physical regimen which forms the basis of sanatorium treatment of pulmonary tuberculosis.

Let us consider the mechanical and physiological action of functional rest of the tuberculous lung. We know from the work of Brauer and Bruns that as a result of functional rest of the lung, the pulmonary circulation is slowed, bringing about a venous hyperemia, the healing value of which has long been established by the classical work of Bier. As a result of lessened respiratory movement, the lymph stream becomes slowed and lymph stasis takes place. Blood and lymph stasis results in lessened toxin absorption through local toxin binding, which causes an inflammatory reaction in the periphery of the disease foci with leukocytic and lymphocytic migration, which is followed by demarcation of the diseased areas and encapsulation with connective tissue.

The favorable results of functional rest obtained by simple recumbency have been a direct incentive to utilize operative procedures for the purpose of giving added rest to the lung, where functional rest obtained through bedrest alone proves insufficient.

A discussion of all the surgical procedures offered from time to time in the treatment of pulmonary tuberculosis is only of historic

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interest. Only those procedures have proved of value which are included under the term "operative collapse therapy" and have for their objective functional rest of the lung obtained in a mechanical way through its collapse. For this purpose we have for consideration only four generally accepted procedures: (1) Artificial pneumothorax; (2) intrapleural pneumolysis; (3) phrenic resection, or exairesis; and (4), extrapleural thoracoplasty. Possibly one might include a fifth, extrapleural pneumolysis with fill.

Artificial Pneumothorax

Of the various operative collapse procedures, artificial pneumothorax has proved, I think, the most valuable, and will therefore be considered first. Statistics on the results of artificial pneumothorax show a striking agreement. The results in 1004 of our cases of partial and complete pneumothorax, covering a period from 1911 to 1926, show 32 per cent clinically well; 20 per cent arrested; 16 per cent unimproved, and 32 per cent died. In cases selected for pneumothorax where no gas could be introduced because of pleuritic adhesions, long observation reveals that only 7 per cent made a clinical recovery as a result of sanatorium methods alone and 55 per cent are dead. These patients were subjected to longer sanatorium care than those where gas was introduced. When one takes into consideration that nearly all were severe forms of tuberculosis (84 per cent, Stage III; 16 per cent, Stage II) subjected to artificial pneumothorax after the failure of other methods, one must admit that the treatment is not only an accepted procedure but an obligatory one in certain cases.

The results of treatment in artificial pneumothorax depend upon many factors: (1) The type of disease, its character, degree of activity, complications, and condition of the opposite lung; (2) the character of the pneumothorax; (3) complications arising during treatment; (4) social and environmental conditions influencing the length of treatment. If social and environmental conditions are good and the patient is willing to coöperate, the results depend upon, first of all and most important, the type of disease; second, the character of the pneumothorax; and third, the condition of the opposite lung.

Importance of Satisfactory Collapse. The value of a satisfactory collapse, as compared with only a partial one, is clearly shown by

our end-results in cases where a satisfactory collapse was established; of these 48 per cent are clinically well; 20 per cent are arrested; 11 per cent are slightly improved or unimproved; and 21 per cent are dead. Whereas, in those cases where only a partial collapse was obtained 13 per cent are clinically well; 13 per cent are arrested; 15 per cent are slightly improved or unimproved; and 50 per cent are dead.

One is strikingly impressed with the value of a satisfactory collapse by the fact that of the clinically well cases in our pneumothorax group (which includes all cases wherein either a partial or satisfactory pneumothorax was established, as well as those cases where no gas could be introduced) 76 per cent had a satisfactory collapse; 18 per cent had a partial pneumothorax, and only 5 per cent were cases where recovery was the result of sanatorium care alone, after attempts to introduce gas were unsuccessful. While the value of a partial collapse is not great, it, neverthless, is of unquestionable value. At the same time, we are quite satisfied that in most cases a partial pneumothorax should be combined with other operative collapse procedures or it should be abandoned and the latter procedures substituted. This would give a 40 or 50 per cent chance of recovery instead of less than a 15 per cent one, which is the best result that can be expected from a partial pneumothorax. However, if other surgical collapse procedures cannot be carried out, a partial collapse is better than none at all.

Influence on Opposite Lung. With reference to the danger of disease in the contralateral lung being activated by a pneumothorax on the other side, our experience has convinced us that this danger has been exaggerated, because improvement in the contralateral lung is often coincident with and actually dependent upon improvement of the pneumothorax side. And while it is, of course, desirable to have the disease confined to one side, this is not the usual type of case coming for pneumothorax treatment. The presence of even active disease in the contralateral lung does not necessarily contraindicate pneumothorax treatment, unless it is extensive or of a rapidly advancing character.

Of much greater importance than the mere presence of disease in the contralateral lung is that the disease on the side to be collapsed be of a favorable type, and that a satisfactory collapse be established.

We feel that disease in the contralateral lung plays a much less

important rôle in estimating the prognosis of a pneumothorax case than the type of disease and the character of the pneumothorax. It may be stated that if recovery takes place, it does so usually regardless of a lesion in the contralateral lung, and if death takes place, it is much more often due to other causes than to progression of disease in the contralateral lung.

The impression that pneumothorax treatment should be withheld until after a thorough trial of sanatorium methods, and that it should be undertaken with much forethought, because of the danger of complications, has slender basis. Less than 2 per cent of our pneumothorax cases died as a result of complications, directly or indirectly related to the pneumothorax treatment, such as gas embolism (2 cases), empyema, spontaneous pneumothorax, etc.

The danger of adhesions preventing a satisfactory collapse increases according to the duration, extent and character of the disease. The longer the duration and the more extensive the process, the less the likelihood of establishing a satisfactory collapse. When one compares the results of treatment of various clinical types of tuberculosis and notes the advantage of a satisfactory collapse, one must come to the conclusion that pneumothorax treatment should be utilized earlier in the course of tuberculosis than is generally customary.

Intrapleural Pneumolysis

Artificial pneumothorax will restore to health from 40 to 50 per cent of cases having chronic and acute forms of tuberculosis; of these cases when treated by the usual sanatorium methods, approximately 7 per cent will recover. Unfortunately, its use is limited, as in approximately 60 per cent of all cases wherein it is indicated, either no gas can be introduced or a satisfactory collapse cannot be obtained because of pleuritic adhesions. The limitations imposed upon the efficacy of a pneumothorax by adhesions is striking. In one group of partial collapse cases, comprising nearly one-third of all our cases selected for pneumothorax, we found that in about 20 per cent a satisfactory collapse of the lung and failure to close cavities was due to the presence of bands, strings and folds of adhesions between the collapsed lung and the chest wall. In approximately one-half of the cases of this type a lung collapse and satisfactory closure of cavities may be obtained by cutting through the adhesion with the galvanocautery under thoracoscopic control,

according to the method of Jacobaeus. We may, therefore, discuss the second operative collapse procedure which is supplementary only to artificial pneumothorax. Namely, the closed intrapleural pneumolysis by means of galvanocauterization of adhesions.

We, ourselves, have had experience with both the Jacobaeus and Unverricht instrument. The Unverricht thorascope has the advantage over the Jacobaeus instrument in that it gives a larger field of vision. However, the instrument is more bulky and frequently the heat is objectionable. The Jacobaeus instrument, on the other hand, is so constructed that either the thoracoscope or cautery may be placed through the cannula after the trocar has been withdrawn; this permits an interchange of instruments before burning, and makes it possible to view the adhesion from two points and burn from the more advantageous one, thus often obviating the necessity of more than two punctures of the chest, which is sometimes necessary with the Unverricht outfit in case the view from the first site of puncture is not satisfactory. Furthermore, there is sufficient space between the cautery shaft and cannula to allow smoke to escape, making it unnecessary to interrupt the operation to ventilate the pneumothorax cavity and free it of smoke during the burning operation.

Table I.—Intrapleural Pneumolysis by Cauterization of Adhesions in Artificial Pneumothorax. Twenty-five Cases.

Results and complications	Type of adhesions: Folds.	Type of adhesions: String and bands.
Technically and clinically unsuccessful—incompletely cauterized	6	
Technically and clinically successful—completely cauterized	5	13
Technically successful—clinical result too soon to report		1
Empyema, tuberculous	4	
Exudate: Slight, not requiring aspiration		10
Exudate: Moderate, requiring aspiration	8	
Hemorrhage—not serious and easily controlled by cautery	1	
Severe reactions—fever, shock, dyspnea	0	0
Severe skin emphysema	0	0

During the past year, we have used this procedure in 25 cases, with the results shown in Table I. Of the 25 cases, 6 were unsuccessful from a clinical and technical standpoint. These 6 cases presented diffuse folds of adhesions which could not with safety be completely cut through, thereby permitting collapse of the lung.

We regard the fold type of adhesions much less suitable for burning than the string and band type. Clinical and technical success followed burning in 18 cases, of which 5 were fold-type adhesions and 13 were of the string and band type. One case is too recent to report clinically, but technically the burning was successful.

We have had no serious accidents. Once we encountered a hemorrhage and 4 cases developed an empyema. Approximately 90 per cent show exudates within twenty-four to forty-eight hours, usually ushered in with a slight or sharp elevation of temperature, which subsides in a few days or a week. The exudate in these cases pursues a course no different from those occurring during the course of artificial pneumothorax, in other words, benign. In no case has it been persistent.

We have burned not only adhesions extending to the lateral aspects of the chest wall, but also apical adhesions, as well as those extending to the diaphragm. It is a simple matter to burn through string or bandlike adhesions extending to the lateral aspects of the chest wall, if they are not too numerous, thus preventing clear view of the cautery. However, apical adhesions are more difficult and must be approached with a greater timidity.

The hemorrhage which occurred in one case should have been avoided. The case presented a broad bandlike adhesion, approximately 4 cm. wide and ½ cm. thick. It was old and well organized and fairly large bloodvessels could be seen on the surface of it. We therefore thrombosed the vessels with a very dull-red cautery and began cutting the adhesion with a colder cautery than usual. No bleeding was encountered, but because of the slowness of burning, the heat of the cautery was increased to hasten burning. The adhesion was cut through; the lung stump remained dry but the chest wall stump oozed some blood which suddenly bled profusely. The bleeding was stopped, however, by the application of the cautery.

In 4 of our cases we have made multiple cauterizations; in 2 instances, four times. Our successes in the band and stringlike types of adhesions have been technically 100 per cent. By that I mean the complete closure of cavities with disappearance of sputum in cases where, in spite of previous high pressures over prolonged periods, it had been impossible to effect a closure or markedly reduce the twenty-four hour quantity of sputum. The following is an illustrative case:

Female, M. M., aged seventeen years, under pneumothorax treatment eight months. In spite of some improvement in the patient's condition, the sputum quantity never diminished below 20 cc. in twenty-four hours. There was a bandlike adhesion roentgenographically demonstrable holding open a cavity. The adhesion was of the apical type. It was burned through in less than five minutes, followed at once by a complete closure of the cavity as the lung collapsed. Within three weeks the patient had no expectoration and her recovery was uninterrupted.

In the case of adhesions in the form of folds with broad bases at the chest wall, then thinning out into what appears a band under the roentgen ray and again spreading out at the lung attachment, our successes technically and clinically have been much less, perhaps 50 per cent. These cases are very puzzling. Upon thoracoscopic examination, one is perfectly amazed at times at the mass of adhesions in the form of folds and shelves with pockets not shown on the roentgen ray plate. In fact, one finds nearly three times as many adhesions upon thoracoscopic examination as are revealed on roentgen ray plates. Naturally, the danger of cutting through folds is apparent as they may contain prolongations of a cavity or large bloodvessels.

The technique of adhesion cauterization is simple. One must, of course, carry out a stricter technique than obtains in pneumothorax treatment. One should orientate oneself on the appearance of the pleural cavity. This can be accomplished by studying a pneumothorax induced in tuberculous cadavers. At the same time, it must be borne in mind that an old pneumothorax in a living person looks quite different than a fresh one in a cadaver. At first, the thoracoscopic picture is confusing. Distance is deceiving. An adhesion which appears to be 1 cm. wide when viewed through the Unverricht instrument, if 5 cm. distant from the lens, appears 5 cm. wide if 1 cm. distant from the lens. Consequently, a considerable amount of practice should be carried out before attempting the procedure. The method is painless, except when one burns near the parietal pleura, and there is no shock.

Our work at the Portland Open Air Sanatorium has been much facilitated by the construction of a surgical unit which contains an inside surgery capable of being made dark, yet thoroughly ventilated and provided with the necessary electrical connections. This room immediately adjoins the roentgen ray room where one can control operative procedures under the roentgen ray.

We are convinced of the value of the Jacobaeus procedure, which we believe will go down in medical history as one of the greatest contributions to pneumothorax therapy. At the same time, we wish to emphasize that we by no means advocate burning all string and bandlike adhesions which may be found upon roentgen-ray examination. In most cases, a satisfactory collapse of the lung can be established in spite of strings and bands by means of stretching them. However, when it can be shown that a satisfactory collapse cannot be obtained, and, in spite of higher pressures, adhesions prevent closure of cavities, then a consideration of the Jacobaeus procedure is obligatory.

Phrenicotomy

Utilizing to its fullest extent intrapleural pneumolysis by means of galvanocauterization as an adjunct to artificial pneumothorax, there is still a balance of about one-half the cases selected for pneumothorax in which no gas can be introduced because of diffuse pleuritic adhesions, or in which only a small pneumothorax can be established, for instance, below, near the diaphragm, which mechanically can effect little or no influence upon the disease higher up, or in which the adhesions are of the multiple-fold type not approachable by means of the thoracoscope and cautery. We have for further consideration in those cases the other operative collapse procedures mentioned, namely, phrenicotomy and thoracoplasty, and possibly, extrapleural pneumolysis.

Section of the phrenic nerve for the purpose of artificially paralyzing the diaphragm was first proposed by Stuertz in 1912. The operation consisted in exposing and cutting out a short section of the phrenic nerve at a point in the neck where it passes over the anterior surface of the scalenus anticus muscle. It has been shown that as a result of the procedure a collapse of the lung is frequently obtained amounting to from one-fourth to one-third its volume. There occur, therefore, to a certain extent, the anatomical and physiological changes associated with a pneumothorax. Stuertz proposed resection of a short piece of the nerve a few centimeters long to prevent its regeneration, but, in spite of this, the hemidiaphragm paralysis was either not complete or not permanent.

Our first experience with this operation was in 1914 when we resected the phrenic nerve, according to the Stuertz method, in some half dozen cases. While in some cases the hemidiaphragm

paralysis was permanent, only one was followed by a satisfactory clinical end-result. In the others, the paralysis was either only temporary or not complete. We, therefore, abandoned the operation until the anatomical studies of Felix and Goetze showed that simple resection of the nerve as proposed by Stuertz was not sufficient because in fully 25 per cent to 35 per cent of all cases, the phrenic nerve, while taking its origin usually from the fourth, and at times from the third and fifth, or all three cervical roots, also received additional fibers from the sixth, seventh and eighth and even the first thoracic nerve. These accessory fibers join the phrenic below the site of resection proposed by Stuertz, and, therefore, continue to innervate the diaphragm in spite of simple phrenicotomy. Felix has found an accessory phrenic nerve arising usually from the fifth cervical root in 25 per cent to 35 per cent of all persons. This accessory branch arises either near or with the subclavian nerve and then separates from it just before the latter enters the subclavian muscle, the accessory branch then continues downward in front of the subclavian vein to join the phrenic nerve just behind the sternal end of the first rib.

In order to interrupt impulses coming from the accessory branches, as well as any communicating cervical sympathetic fibers of the phrenic nerve, Felix proposed an exairesis of the phrenic nerve after cutting it, by winding it around a Thiersch, or hemostatic forceps. By this method, at times the entire nerve may be extracted. We have one such specimen, showing even the ramifications upon the diaphragm. One may feel confident that complete interruption of the phrenic and associate fibers has taken place, if 12 cm. of the nerve is evulsed. We have never seen a failure when even shorter sections (8 to 10 cm.) have been drawn out.

Goetze overcomes accessory impulses by resecting the subclavian nerve and all accessory fibers between the phrenic and cervical nerves. While the "phrenico-exairesis" as Felix designates his operation, is the procedure today most generally carried out, it has certain dangers, and there can be no doubt, in view of more recent anatomical studies on the phrenic nerve, that the Goetze radical phrenicotomy is the operation of choice in certain cases.

In collaboration with Dr. Andreas Plenk, assistant in the surgical division of the Jubilienspital, Vienna, we dissected out 112 phrenic nerves in cadavers. We found 72 per cent typical phrenic nerves, that is to say, the nerve arose from fibers either from the third, fourth

or fifth cervical nerves which joined together in one main trunk and passed down across the anterior surface of the scalenus anticus muscle and entered the thorax behind the subclavian vein.

In 32 instances, or 28 per cent, the formation of the nerve was atypical. Double phrenics with associate fibers on the face of the scalenus anticus were encountered 8 times. In 4 instances the associate phrenic came from the subclavian nerve. In 1 instance there was a bilateral and in 1 instance a unilateral absence of typical phrenic, there being no main trunk on the anterior surface of the scalenus anticus muscle, but two large trunks coming mostly from the fifth cervical close to the point of origin of the subclavian nerve. They should really be called "associate phrenics" as all three passed down in front of the subclavian vein. These latter three nerves came off so far lateralward that they could not be located by the technique of Felix or by the usual surgical approach for a phrenicotomy.

It was probably just such a case which Sauerbruch encountered and failed to find the phrenic nerve. We have met with 2 such cases in practice where no phrenic nerve could be located on the anterior surface of the scalenus anticus muscle however, it was found far out in combination with the subclavian nerve. In both instances, resection of the nerve produced a perfect hemidiaphragm paralysis.

In 23 instances, the accessory phrenic fibers came in common with the subclavian nerve from the fifth cervical. In 6 instances, the accessory phrenic arose apart from the subclavian nerve as a separate branch. In 5 instances, it formed a loop around the vein. In 1 instance, two associate phrenic trunks passed in front of the subclavian vein, then looped around behind the internal mammary artery.

From these studies, it would seem that in case of a typical phrenic nerve, the exairesis of Felix is adapted, but even then, not without possible danger.

While it is reported in the literature that the phrenic nerve sometimes passes through the subclavian vein, we have never seen it, but we can confirm Goetze's observations regarding the intimacy between the associate phrenic nerve and the vein. In one instance we found the associate phrenic passing through the wall of the subclavian vein. An exairesis in such a case would probably lead to severe, if not fatal, bleeding. Fortunately, this type is not common. Friederick encountered a phrenic nerve intimately bound to the

internal jugular vein. During exairesis, the wall of the vein was torn and a fatal air embolism took place.

In the complex types of phrenic nerve, the radical phrenicotomy of Goetze alone appears practicable. Whether an exairesis, or radical resection, is the operation of choice, will depend entirely upon the type of phrenic met with at time of operation. In our operated cases, now numbering 90 phrenicotomies, we have met with abnormalities in about 30 per cent of them. Based upon these anatomical studies, our surgical approach is through the subclavian triangle because of the accessibility to the associate fibers.

We make an incision 2 to 3 cm. long, 2 cm. above and parallel with the clavicle, extending from the posterior border of the sterno-cleido-mastoid muscle lateralward, and enter the subclavian triangle below the omohyoid muscle. The platysma muscle, superficial and deep fascia are divided. The external jugular vein is either ligated and cut or retracted. The pyramidal fat body, together with the small lymph glands usually met with, are retracted. Below will be seen the transverse scapular artery, and above, the superficial cervical artery, both of which cross the phrenic nerve transversely in front of it. The phrenic nerve is sought out on the anterior surface of the scalenus anticus muscle, separated from the fascia and picked up with the tenaculum. After positive identification and further examination of the field for associate branches (for which purpose the incision may be lengthened), the nerve is injected with novocaine and resected. In case no complex types are met with, an exairesis is done. If, on the other hand, associate fibers are found, these are cut and then an exairesis of the main stem is performed. We feel that an exairesis is perfectly safe under those conditions.

The end-results in our series of 90 cases where a phrenicotomy was performed, are shown in Table II.

The most striking benefit followed phrenicotomy in the fibro-caseous cavernous group, stationary or slowly progressive, and in good general condition with a good contralateral lung. As a matter of fact, in this group, 5 patients are clinically well as a result of the phrenicotomy alone. Each of these cases had demonstrable cavity formation in an upper lobe.

Our experience, particularly in this group of cases, supports our contention that a phrenicotomy should precede every thoracoplasty. We have also convinced ourselves of the value of a phrenicotomy with a partial pneumothorax in selected cases.

TABLE II.—RESULTS OF PHRENICOTOMY

Type of case	No. cases.	General condition. Good.	Fair.	Bad.	Unimproved.	Improved	Much improved	Clinically well.
Fibrocaseous cavernous tuberculosis (unilateral); stationary or slowly progressive, thoracoplasty indicated	40	35	3	2	7	21	7	5
Bilateral productive tuberculosis (one side extensive); pneumothorax impossible, thoracoplasty contraindicated; phrenicotomy on worse side	17*	8	4	5	4	10	3	
Exudative forms of tuberculosis; pneumothorax impossible; thoracoplasty contraindicated	9†	.		9	9			
Supplementary to artificial pneumothorax	8	5	3	..	4	3	1	
After pneumothorax prior to thoracoplasty; expanding lung; reactivation of disease	6	4	2	..	2	2	2	
Bilateral cirrhotic cavernous tuberculosis stationary cavity contralateral lung; phrenicotomy worse side; pneumothorax impossible	4	2	1	1	1	1	2	
After thoracoplasty for additional collapse	1	1	..	..	.	1		
Bronchiectasis	5	5	..	..	.	..	2	3
Total	90	60	13	17	27 (30%)	38 (42.2%)	17 (18.8%)	8 (8.8%)

* One developed pleurisy with effusion opposite side.
† Two developed exhaustion psychoses.

Our results in exudative types of tuberculosis have been unsatisfactory. In no instance could we see that it altered the progress of the disease or influenced it in a favorable manner. Two of our patients in this group developed a toxic exhaustion psychosis after the phrenicotomy. This may have been precipitated by apprehension or possibly the pain of exairesis, as in both cases the exairesis was painful. In one case the psychosis lasted one month, the patient's mental state then becoming normal, but the disease was uninfluenced. In the other case, the psychosis continued until death.

In cases where the diaphragm pursues a horizontal course and is fixed by dense pleuro-diaphragmatic adhesions, a phrenicotomy is of no value. We cannot agree with those who maintain that a phrenicotomy is of no value in apical lesions. We have convincing evidence of its favorable influence upon cavities situated in the apex, and we believe the benefits of a phrenicotomy are due not only to the rest given the lower lobe but also largely to the reduction

in lung volume. Especially marked are the results in cases where dense parietal adhesions prevent intercostal separation during inspiration and only abdominal breathing exists. A phrenicotomy in these cases results in complete rest to the lung.

Because of its simplicity and great value, a phrenicotomy must be considered next after a pneumothorax and before other more radical procedures. We believe that a phrenicotomy should be resorted to first in every case before a thoracoplasty, because occasionally so much benefit is seen from a phrenicotomy that the more radical procedures, such as extrapleural pneumolysis or thoracoplasty, are unnecessary. Besides, a phrenicotomy serves as a test of the integrity of the contralateral lung in case it also presents evidence of disease. While failure of any disease in the contralateral lung to become active and progressive following a phrenicotomy does not give positive assurance that a more radical operation will not be followed by such changes, we can feel confident that, on the other hand, if any disease in the contralateral lung becomes active or progressive following a phrenicotomy, more radical procedures are contraindicated. Accordingly, we precede every thoracoplasty by a phrenicotomy, and in at least five instances of contemplated thoracoplasties, so much improvement followed a phrenicotomy that further operative procedures have been considered unnecessary.

A phrenicotomy may be done in bilateral cases on the worse side if one has become convinced that the worse side is mostly the source of constitutional disturbance. If the cases have been correctly chosen, improvement in cough and expectoration should take place following phrenicotomy. The reduction of cough and sputum quantity following a phrenicotomy is sometimes extraordinary. Hemidiaphragm paralysis, instead of making expectoration more difficult, renders it more easy. A perfect paralysis of the diaphragm must be accompanied by a high position and no movement, or, at least, only a paradox one. There should be a positive Bittdorf and Hitzenberger phenomenon.

Phrenic resection or exairesis is not without danger. Sauerbruch reports several cases which he had opportunity to examine after they had been operated upon in other clinics, and found that instead of the phrenic the vagus nerve had been cut. We have encountered one case wherein the phrenic was supposedly cut. We found, however, a negative Bittdorf and Hitzenberger phenomenon and normal movement. We thereupon reoperated the case and found the

phrenic nerve lateral to the scalenus anticus muscle. It seems probable that this was a case presenting a double phrenic.

Sauerbruch has also had several cases of air embolism as a result of opening large throat veins, but no death took place. Once a Horner symptom-complex was observed following injury to the sympathetic. The thyrocervical axis can become a source of much concern if opened close to the subclavian artery, as the control of bleeding is then very difficult. Sauerbruch was twice compelled to ligate the subclavian artery, in spite of which one case was fatal. The possibility of mediastinal suppuration must be considered, although it is not a common complication. We once evulsed a phrenic nerve to which was attached a portion of an anthracotic hilus gland. Other less serious accidents may occur, and while these accidents are not common, an exairesis or resection is not a procedure to be regarded as strictly trivial. It should not be attempted without a thorough knowledge of the topographical anatomy of the neck, and, particularly, familiarity with the abnormalities of the phrenic nerve. Qualified assistants should be available, as should also preparations to meet any emergency which might arise.

As a single procedure in selected cases and as a supporting operation we are convinced of the value of phrenicotomy. As a matter of fact, as shown in Table II, of 54 cases where a thoracoplasty was indicated and decided upon either after failure to establish a pneumothorax or where the lung had reëxpanded following a pneumothorax, 5, or 9.2 per cent made a clinical recovery as a result of the preliminary phrenicotomy. We believe that a phrenicotomy should precede every thoracoplasty, but we do not feel that it is justifiable or necessary to do a phrenicotomy in combination with every pneumothorax, irrespective of whether the collapse is satisfactory or not.

Extrapleural Thoracoplasty

Next to a satisfactory pneumothorax, an extrapleural thoracoplasty is the most valuable procedure for placing the lung at rest. After many modifications in technique, credit for giving us a surgical procedure of unquestionable value and low mortality in properly selected cases belongs to Brauer and Sauerbruch, whose methods are the two most generally employed. Both have undergone so many modifications that, at present, little difference exists between them. Sauerbruch emphasizes the importance of a vertical collapse and

resects the ribs close to the transverse processes. Brauer also now recommends cutting the ribs close to the spine. The importance of removing the ribs close to the spine cannot be too strongly emphasized as the degree of collapse after a thoracoplasty depends largely upon the removal of that portion of the ribs between the angle and tubercle, over the costovertebral gutter. Sauerbruch formerly resected 7 to 8 cm. of rib, but now does not hold himself to any certain length. If some fibrosis of the lung is present, he always removes 8 cm., but if little fibrosis of the lung is present, he resects 12 cm. and even more. The underlying pathology also determines how much rib is resected and whether the eleventh is left or not, but he always takes out the first. Brauer takes out from 12 to 16 cm. and more, in order to permit the scapula to lie deep in the thorax like a cushion in the resected place. In fact, the sign of a well performed thoracoplasty is to be able to demonstrate the vertebral border of the scapula lying in front of the vertebral ends of the resected ribs. To accomplish this, Brauer resects somewhat more rib than Sauerbruch, however, those who have had wide experience, agree with Brauer that this is important. The Brauer method is called "subscapular-paravertebral resection" and differs very much from the old Brauer-Friedreich operation, where long sections of ribs were removed in the lateral wall of the chest, leaving a flaccid chest wall thinly covered with muscles, which did not become fixed by new bone formation. Consequently, the chest wall swung in and out with inspiration and expiration in paradox movement, or so-called "pendulum respiration," the same movement being shared in by the mediastinum, unless it was fixed by adhesions. And, in addition to the graver danger from the standpoint of operative shock, the old Brauer-Friedreich operation did not always give the desired rest to the lung. The new Brauer operation is practically the same now as the Sauerbruch, except that Brauer resects longer sections of ribs beneath the scapula. Brauer does not remove the eleventh rib, and removes the first only when the apex shows no retraction. This operation gives a greater collapse than the Sauerbruch and there is very little chest deformity and no chest flutter.

It has been our policy to operate under local anesthesia, unless the patient is raising small quantities of sputum or is very apprehensive, in which case we employ gas anesthesia or a combination of local with gas analgesia.

In connection with the anesthesia of the intercostal nerves, great

care should be exercised not to inject too close to the vertebral column. We have been able to demonstrate that dyes injected in proximity to the intercostal nerves, 6 cm. from the mid-vertebral line, may pass along the perineural lymph spaces for some distance from the point of injection. We have found it in the sheath of the rami communicantes, extending sometimes to the sympathetic ganglia, also in the spinal dura, and it seems therefore evident that novocaine may also pass along the perineural lymph spaces to the spinal dura with considerable ease, and it is easy to understand that toxic symptoms from novocaine, instead of being due to "hypersensitiveness," may be caused by the rapid absorption of the anesthetic by the spinal dura or its absorption by the sympathetic ganglia.

We have no fixed rule regarding the sequences of the two-stage thoracoplasty, nor the amount of rib we remove, being governed entirely by the underlying lung pathology. We believe the matter of sequence of the second stage thoracoplasty should be individualized. We do a preliminary phrenicotomy in every case for the reason stated under phrenicotomy. Usually the operation can be carried out quickly in either stage. We always resect the first rib but have no fixed rule regarding the eleventh. The lower six ribs, excluding the twelfth, which is never disturbed, should be removed and the wound closed in twenty to thirty minutes, and the operation should never exceed forty minutes for resecting the remaining upper five ribs.

Upon the after-care depends, to a great extent, the patient's fate. The most important thing is to give sufficient opiate to relieve the pain of coughing and then encourage the patient to cough and drain the lungs of sputum about every four hours, unless the case is one having little sputum. For this purpose a nurse should be in constant attendance, to support the operated side.

The operation should never be done in one stage but always in two or more stages, depending upon the patient's condition. Argument that the operation should be done in one stage because the patient would never return for the second, after having been through one ordeal, is not in accordance with our experience, as none of our cases have refused the second operation or even a third. Moreover, the mortality is tremendously lessened by doing a two-stage operation, as Brunner has shown, from 27 per cent in the one-stage to 4 per cent mortality in the two-stage operation.

Furthermore, the collapse is just as good in a two-stage operation, providing the second stage is done within a reasonable time after the first, before new bone formation takes place. Aside from the lessened danger of shock in a two-stage operation, there is also less danger of activating disease in the other lung.

Before the second stage is attempted, a careful physical and roentgenological examination must be made for the purpose of determining whether any changes have taken place in the contralateral lung which contraindicate further operative procedures. The upper phase of the operation is more difficult than the lower because of the position of the first rib, depth of the shoulder muscles, presence of the scapula, and difficulty of obtaining a good anesthesia. Nevertheless, a section of the first rib should invariably be removed, unless definite apical retraction has taken place.

A complete thoracoplasty is indicated in pulmonary tuberculosis with, or without, cavity formation when the disease is essentially unilateral and of a productive type, providing the patient is in good general condition and not too old, and providing attempts to establish a pneumothorax have failed, or if adhesions prevent the establishment of a satisfactory collapse, and the partial collapse is of such a character that a recovery is considered improbable. While the best results will be obtained in the fibroid or productive types, the exudative types give the most unfavorable results. The presence of large cavities also render the prognosis less favorable.

The more severe unilateral exudative forms of pulmonary tuberculosis are rarely suited to a thoracoplasty and will not be productive of good results. In rapidly progressive, acute forms, a thoracoplasty is contraindicated. The contraindications for a thoracoplasty otherwise are practically the same as for a pneumothorax, except that the presence of an active lesion in the contralateral lung is a contraindication, unless it is of minimum extent and certainly not progressive. Decision regarding the significance of a lesion in the contralateral lung must be decided in each case, after careful examination upon its character, extent, and degree of activity. The results in our series of 40 thoracoplasties are shown in Table III.

The results of a good thoracoplasty are striking; clinical improvement follows after recovery from the effects of the operation. The cough and sputum quantity diminish constantly. The temperature, which before the operation may have been unstable, pursues a

normal course and the patient's general condition improves parallel with the clinical improvement.

TABLE III.—RESULTS OF EXTRAPLEURAL THORACOPLASTY

Number of thoracoplasties	40			
Dead directly due to operation	1	2.5%	5%, remote operative mortality.	
Dead indirectly due to operation	1	2.5%		
Dead from causes not due to operation	2			
Worse	1			
Unchanged	4			
Somewhat improved (recent cases)	15			
Greatly improved (less than 3 months since operation)	10	25%	40%	Improved and well group; clinical recovery probable in entire group.
Clinically well	6	15%		

The good results of a good thoracoplasty in properly selected cases show approximately the same percentage of recovery as a satisfactory pneumothorax, or, about 40 per cent clinical recoveries, 30 per cent improvement, and 30 per cent early and late deaths. Some thoracoplastic surgeons, as Sauerbruch, have gone so far as to consider a thoracoplasty a more valuable operation than a pneumothorax. It is true that a thoracoplastic collapse has an advantage over a pneumothorax in that it is unchanging, whereas in a pneumothorax, the degree of collapse or compression changes with gas introduction and absorption, or, as a result of an obliterative pneumothorax, the lung expands in spite of efforts to keep it collapsed. Another advantage of a thoracoplasty is that the operative procedure is over in two sittings as a rule, whereas with pneumothorax treatment, the refills must be kept up for years. Therefore, it is true that pneumothorax treatment is long, yet it has the advantage that the patient is kept under control. Arguments that pneumothorax patients abandon treatment because of its duration, and that a thoracoplasty is better because "once done always done," and the patient cannot abandon treatment, is nonsense, because one who will abandon pneumothorax treatment will also abandon the after-care so essential for success following a thoracoplasty.

That cases presenting adhesions interfering with collapse of the lung should have a thoracoplasty because stretching adhesions by increasing the intrapleural pressure or burning them according to the Jacobaeus procedure is more dangerous than a thoracoplasty, can only represent the expression of opinion of one who lacks both experience in pneumothorax and with the method of Jacobaeus. We have been repeatedly successful in stretching or separating and

burning adhesions and establishing a satisfactory collapse, and have never had a serious accident. Of course, proper judgment must be exercised in using high pressures when dealing with adhesions, as too rapid separation may result in gas embolism, empyema or spontaneous pneumothorax. However, the danger of complications in pneumothorax cannot be compared with those of a thoracoplasty. Dissemination of disease to the contralateral lung or its activation, if already there, is not a frequent or dangerous complication in pneumothorax, but it is common and dangerous after a thoracoplasty, as from 10 to 15 per cent develop progression of disease in the contralateral lung. Consequently, one cannot be so liberal regarding either the presence of disease in the contralateral lung, or its activity in considering a thoracoplasty. Moreover, a thoracoplastic collapse is never as good as a satisfactory pneumothorax. Besides, it is unnecessary to subject a patient to a severe surgical procedure with a high operative risk of from 5 to 15 per cent, and permanently put a lung out of function, when a pneumothorax will give a better result, and has nearly no operative risk. Furthermore, after the pneumothorax is discontinued, there invariably is a functionating lung which may be the means of saving the patient's life, in case of pneumonia.

At the same time there can be no doubt, providing the case is suited and the operator qualified, that a thoracoplasty should be advised in appropriate cases, as certainly the danger of the operation is much less than the danger of harboring a chronic pulmonary tuberculosis.

The general impression has been that these operative collapse procedures come into question only for a relatively few cases. During the first twelve years of our pneumothorax experience, we subjected 10 per cent of our cases to that form of treatment, but as a result of our studies we now adopt operative collapse therapy in about 20 per cent of our sanatorium cases, of which about 25 per cent are Stage II and 75 per cent are Stage III cases. Of these cases, an artificial pneumothorax will prove satisfactory in about 40 per cent. Of the remaining cases, adhesions will prevent any gas introduction in one-third, and in two-thirds adhesions will prevent a satisfactory collapse. Of this latter group, about 20 per cent will be suited for the Jacobaeus procedure and in one-half of them it will be successful in establishing a satisfactory pneumothorax. Thus, artificial pneumothorax alone, or combined with an

intrapleural pneumolysis, will be successful in about one-half the cases in which it is indicated. Of the remaining half, where either no gas could be introduced or a satisfactory pneumothorax is impossible because of adhesions not approachable by the Jacobaeus procedure, a phrenicotomy will be indicated in most of them and about 9 per cent will recover. A thoracoplasty will be indicated in about one-fifth of the cases selected for pneumothorax where the latter procedure cannot be carried out—of these about 40 per cent will recover.

The number of cases that should be treated by operative collapse procedures is higher than one would suppose. We feel sure that had we utilized operative collapse procedures to the extent we now think indicated, many of our cases now dead would be alive and rehabilitated to useful lives.

These special surgical procedures from a pure surgical standpoint are not difficult. At the same time, they do require mechanical skill and knowledge of topographical anatomy as well as general surgical training and experience. A surgeon who is not well versed in thoracoplastic surgery or has not an intimate knowledge of the technique, had better avoid attempting the operation.

The selection of cases, the before- and after-operative treatment are so important that tuberculosis specialists and surgeons should, if possible, spend some time in a hospital or sanatorium prepared to teach this special branch of surgery. Undoubtedly, the time will come when the tuberculosis specialist, in addition to mastering the complicated nonsurgical methods of treating tuberculosis, must also be qualified to do the other operative-collapse procedures besides pneumothorax, and thus have command over all the therapeutic measures indicated in this disease.

Note.—The cases upon which these observations are based represent those on the combined service of myself and my associates, Dr. Ray W. Matson and Dr. Marr Bisaillon, at the Portland Open Air Sanatorium.

DISCUSSION

Dr. A. P. C. Ashhurst: The methods of treatment described by Dr. Matson appeal to me because each one of them is a mechanical treatment for the cure of disease; and this I believe to be the best form of surgery. When we attempt by mechanical means to alter chemical processes, or to affect the sympathetic nervous system, we will soon—but sometimes not soon enough

—discover our errors, because no one yet knows enough of chemical processes or of the sympathetic nervous system. Artificial pneumothorax, as well as the method of Jacobaeus, and extrapleural thoracoplasty are all of them good sensible surgical procedures because the indication to be met is a mechanical one, namely, to secure compression of the lung. I am interested to hear that Dr. Matson has had such good results from his long-continued artificial pneumothorax, because the impression of the general practitioner seems to be that this is scarcely worth trying and might as well be abandoned in favor of thoracoplasty; and I have had the personal experience of having the family physician cast reflections on my up-to-date-ness because of my suggestion that artificial pneumothorax be tried before thoracoplasty.

I would like to ask Dr. Matson if he has had any trouble with regeneration of ribs; he mentioned it in one case; and if he has had this trouble, whether he has used any means of preventing such regeneration by the application of Zenker's solution to the rib bed at the time of operation?

I would also like to know where I can get one of the periosteal elevators he mentions, which works backward and forward at the same time.

Dr. John B. Flick (by invitation): It is with great pleasure and much profit that I have listened to Dr. Matson's paper. He speaks with the authority of a ripe experience in the treatment of tuberculosis by compression therapy, both medical and surgical.

The benefit of compression of the lung by means of artificial pneumothorax is now well known to the medical profession and pretty well accepted as a successful method of treating selected cases of pulmonary tuberculosis. But the possibility of compression by means of a plastic operation on the chest wall is not generally recognized. It is a subject which deserves the attention of both the internist and the surgeon. The mere mastery of technic is not sufficient in this type of surgery, where a knowledge of the disease and the principles which govern its treatment are equally if not more important. While the selection of cases primarily falls to the internist, the responsibility for operation rests on the surgeon; and he should be qualified to pass judgment on their suitability.

The monograph of John Alexander on "The Surgery of Pulmonary Tuberculosis," the work of Gravesen which has been translated into English, and the many good articles which have appeared in the current medical literature of this country in recent years, are beginning to awaken widespread interest.

My experience with phrenicotomy has been very small. I have performed this operation on but 2 cases. One apparently received no benefit, the other was slightly improved and later I did a paravertebral thoracoplasty. Both of these cases were borderline, and the phrenic nerve was divided as a therapeutic test, as advocated by Sauerbruch. Both of these cases afterward were studied with the fluoroscope and showed elevation of the diaphragm on the affected side, but the diaphragm still moved and in a paradoxical fashion. The patient who apparently improved and upon whom I did a first-stage paravertebral thoracoplasty, taking out only five ribs, died eleven days after operation. Autopsy failed to disclose the cause of

death, but I believe that it was from exhaustion and that my judgment was poor in accepting him for operation.

Two of our most striking results have been in cases complicated by a tuberculous empyema, following pneumothorax treatment. The first was operated upon about two years ago, the second about six months ago. Neither has required aspiration since the final stage of a paravertebral thoracoplasty.

I have had no experience with apicolysis, but would prefer, where large apical cavities exist, to do a paravertebral thoracoplasty, with removal of further sections of the upper ribs through an axillary incision, as advocated by Welles, of Saranac Lake.

The division of pleural adhesions, which prevent successful pneumothorax treatment, by means of a galvanic cautery, through a thoracoscope as practised by Jacobaeus, does not appeal to the average surgical mind. The results that Dr. Matson and others have obtained, however, would seem to justify its employment.

I prefer local infiltration anesthesia with nitrous-oxide oxygen analgesia. The anesthetic is not carried deep enough to abolish the cough reflex. With nitrous-oxide analgesia, the patient, I believe, is less shocked and both patient and surgeon are more comfortable.

The dictum has been handed down to us, like the old idea of the incurability of tuberculosis, that surgery in tuberculosis is a thing to be avoided, and that these patients do not stand operations well. Yet those of experience know that, with good judgment in the selection of cases and an operation in stages, paravertebral thoracoplasty has a reasonably low mortality and will restore to a useful existence many cases of pulmonary tuberculosis which otherwise would be doomed, if not to death, at least to a life of invalidism.

Let us hope that Dr. Matson's paper will stimulate interest in his hearers equal in measure to the excellency of his work.

Dr. Henry R. Landis: I think there is one point which Dr. Matson failed to make clear, and that is that much of the success in carrying out special therapeutic measures is due to teamwork. There should be close association between the internist and the surgeon. I never expected to hear a surgeon confess that his duties were largely mechanical. I believe that in this particular line of surgery the surgeon has to know as much about the disease as does the internist. It is rather curious that Dr. Matson started his career as a bacteriologist; he then specialized in internal medicine and eventually as a tuberculosis specialist, and at a relatively recent date he has undertaken this rather formidable type of surgery. I think a large part of his success in surgery is because of his thorough and complete knowledge of the disease. Of course, an ideal way is with the team of internist and surgeon working together, and I know of such a team. In this way the surgeon acquires an excellent knowledge of the disease with which he has to deal. One man does the mechanical work and the other man is responsible for the indications; but I think the greatest success will always be attained by the man who knows both sides pretty thoroughly.

PRESIDENT: Many years ago there was a very common idea in the profession that if the phrenic nerves were severed this resulted in paralysis of the diaphragm and death. In the Cartwright Prize Essay of the College of Physicians and Surgeons of Columbia University in 1889, there are records to show that phrenic nerves can be cut in every animal from the guinea-pig to the dray horse, not excluding the monkey—with no great discomfort to the animals. Therefore phrenic section, in animals at least, does not possess the dangers that it was supposed to have possessed in years gone by.

DR. MATSON (closing): Regarding the possibility of thoracoplasty replacing pneumothorax; I cannot conceive of such a situation. Sauerbruch, some four years ago, intimated such a thing. Nevertheless, a thoracoplasty is never justifiable when a pneumothorax can be done or until a pneumothorax has been attempted. A satisfactory pneumothorax will result in recovery in 40 to 50 per cent of all cases; results certainly superior to thoracoplasty. At the same time, after the lung has reëxpanded following a pneumothorax, one has a good functionating organ, which, among other things, is a protection in case of acute respiratory infections such as pneumonia, for instance. This complication in a thoracoplastic case is invariably fatal, whereas in pneumothorax, with two good lungs, the mortality is no greater than in uncollapsed cases. Furthermore, the direct and indirect mortality in pneumothorax is less than 2 per cent. This applies to empyema, spontaneous pneumothorax, gas embolism and so forth. In thoracoplasty the operative mortality is much higher. It is unjustifiable, therefore, to subject a patient to a severe procedure, such as thoracoplasty, provided an artificial pneumothorax can be done.

Regarding the Jacobaeus work, I consider it one of the greatest contributions to the treatment of pulmonary tuberculosis since pneumothorax was brought out. The Jacobaeus operation is not as difficult as we are led to believe. With the modern Unverricht instrument one gets a beautiful view of the pneumothorax cavity, and there can be little doubt regarding the nature of tissues about to be burned. That the operation is an extremely dangerous one, I am sure is not according to the experience of those who have been working with modern instruments in properly selected cases. With the old type of Jacobaeus instrument, I regard the operation a dangerous procedure.

As to Dr. Ashhurst's question concerning rib regeneration, I would say that this gives us no trouble and we do nothing to prevent it; in fact, we desire rib regeneration after a complete collapse has been produced. We do strive to get out sufficient rib, so that when it does regenerate we do not have to dig out newly formed rib, as this is a most difficult procedure.

The instrument which I have designed is made by George Tieman & Company, New York. I would be very glad to give Dr. Ashhurst one of the instruments I described and have him try it out.

APPENDIX

INTRODUCTORY NOTE TO THE SECTIONS

Almost all of the papers read before the various sections of the College are subsequently published in full in medical periodicals.

The papers listed in the following pages may be found by consulting the *Index Medicus*, provided a sufficient length of time has elapsed from the date of publication to the next succeeding quarterly number of the *Index Medicus*.—[Editor.]

PROCEEDINGS

OF THE

SECTION ON OPHTHALMOLOGY

JANUARY, 1927, TO DECEMBER, 1927

JANUARY 20

Reconstruction of the Eyelids by Epithelial Grafts

Dr. C. E. G. Shannon and Dr. George H. Cross presented a case of cicatricial ectropion of all four lids with plastic reconstruction by the epithelial outlay method of Gillies. The patient, a young married woman, had suffered severe burns of the lids and face in November, 1923, while polishing a stove with polish that contained benzine. Exposure keratitis, fortunately, has not developed.

The operation was carried out first on the right side, and, two weeks later, on the left. An incision was made about 3 to 4 mm. from the line of the imbedded lid margin, extending from canthus to canthus, and separation made in what may be designated as the lines of cleavage. Following the dissection, a Thiersch graft, taken from the inner side of the thigh, was placed with the raw surface outward over a mold of modeling compound and imbedded in the wound, and the wound edges brought together with sutures. Ten days later, the mold was removed by cutting with scissors along the line of incision, and removing the mold with forceps. At first there was quite a depression at the site of the graft on either side, but, in the course of time, aided by massage, these depressions gradually filled out.

In connection with this interesting operation, certain important facts should be borne in mind, as emphasized by Gillies:

1. Operation should not be considered until contraction of the tissues to be grafted has entirely ceased, otherwise the graft will develop unevenness from shrinkage of the structures beneath.

2. That the graft completely covers the mold before insertion into the wound.

3. That infection may supervene if excess of graft is not removed.

4. Particular care in the removal of the graft should be taken in opening up with the scissors the areas of the wound in which the mold was buried. If this is not done, the areas are apt to be pocketed.

Dr. George H. Cross pointed out that the Gillies outlay is a modification of the Essers inlay. The grafts are wrapped around molds of modeling compound with the outer surface next the mold. They are then buried in the tissue they are to cover and the edges of the wound sutured together. He showed slides depicting the results of the operation in the various stages of the case reported.

Dr. Cross explained the differences between the inlay, outlay and overlay, as applied to skin grafting, in the correction of defects of the eyelids. A case of contracted orbit, in which the skin was undermined with a scissors through a vertical incision just below the outer canthus, was shown, and by a drawing he illustrated the method of producing this pocket and showed the mold which was to be covered with an epidermal graft and inserted. At the end of about seven or eight days after insertion, an incision is made through the conjunctival surface of the orbit down through the graft to free it from the mold; the mold, being removed from the orbit, leaves a well-lined lower sulcus. This is different from the outlay, where the mold is removed through the same incision in which it was inserted.

Four other slides illustrated the overlay method of correction of cicatricial ectropion of three eyelids of a man burned with sodamide, a compound used in the manufacture of indigo. Here, following the removal of dense scars and the preparation of the lids for the graft, a thin epidermal graft was placed in position, then covered with a layer of warm compound, over which a tight bandage was applied. This enabled a firm, even pressure to be maintained to all parts of the graft. There should be no undue hurry to remove the original dressing. The longer it is allowed to remain, the more likelihood of a perfect take. This is different from the outlay in that no suturing is required, and later inspection would show how little difference there is in the color of the grafted area compared with the surrounding surface.

Dr. Cross also showed three slides of a case operated upon by the outlay method, giving a better idea of the steps of the operation. He pointed out that it is most essential that the graft be as thin

as tissue paper, if such a comparison may be used. It may then be molded with the fingers so that it will cover the mold and will stay in place without suturing. Grafts that curl up and refuse to stick to the mold are too thick.

Finally, slides illustrating the older, open method of skin grafting were shown. In this method nothing was allowed to touch the graft during the healing process.

Thus he showed three different methods for the correction of the type of lid defect of which Dr. Shannon's case was an example. In his case the outlay was chosen with the feeling that it would give the best results. He was inclined to think that if a similar case would present itself again he would try the overlay method. Nevertheless, it is possible to reach a desired result by several different routes or methods. Dr. Shannon and Dr. Cross both felt very much pleased and gratified with the results as obtained in this case.

Discussion. Dr. William Zentmayer stated that he had a single experience with this procedure and was likewise assisted in the operation by Dr. Cross. It was a case of cicatrical ectropion of the lower lid from a torch burn. The immediate result was disappointing in appearance, although the ectropion was corrected. The surface was very uneven. This, however, disappeared in the course of time.

The Principles of Syphilotherapy as Applied to the Eye

Dr. John H. Stokes stated that the effective treatment of syphilis suffers today from a variety of defects, all more or less inevitable in the growth of the specialty of syphilology from the diffuse half-knowledge of the clinical era of the ninteenth century. Traditional syphilotherapy was regional, while the modern syphilologist envisages and effectively manages the entire disease in any individual patient, rather than any one particular lesion. By the same sign, the point of view of specialists in other fields is apt to be local and restricted.

Ophthalmologists have labored under difficulties in their effort to deal with syphilis of the eye in modern times. The eye mirrors the nervous system, is the least accessible to influence of treatment and is the scene of some of the most critical forms of relapse, especially under inadequate treatment. Moreover, since Ehrlich's warning against the possibility of unfavorable effect of the use of arsenicals on the optic nerve, it is small wonder that eye men have been conservative. Likewise, as in syphilis of other organs, one should

not expect treatment to do away with scars, such as the Argyll-Robertson pupil.

Two Fundamental Principles. First, treatment should not be begun until the entire condition of the patient has been evaluated by complete examination. Second, syphilis of a special structure should be treated as systemic rather than local syphilis.

Three Characteristics of Arsphenamine Therapy. First, it produces a flare-up when first given, which may be damaging. Second, it is responsible for fulminating relapse if stopped too soon. Third, it produces healing with fibrosis too rapidly for functional adaptation. Therapeutic shock is less frequent and serious in the eye, but it may be disastrous. So the patient should have preparation with slower-acting drugs and graded arsphenamine dosage.

Relapse and Allergy. Arsphenamine causes a wholesale destruction of spirochetes, and may render the patient "allergic" as well as physiologically defenseless by the reduction of their numbers, for the drug removes the very excitant of bodily resistance.

The Therapeutic Paradox. This term means essentially damage by fibrosis in too rapid healing. There is no reason why a similar effect should not be produced in the eye, as well as in the heart and liver, by the injudicious use of arsphenamines.

Specific vs. Nonspecific Treatment Effect. Treatment for syphilis, irrespective of its action on the Spirocheta pallida, has healing effects through stimulation of general defense. This is shown in its effect on other disease processes. The effect of a false therapeutic test may be produced by a wrong interpretation of nonspecific effects.

Intensive Treatment without Therapeutic Shock. Arsphenamine, because of its attack on the Spirocheta pallida, is responsible for the therapeutic shock; mercury stimulates cellular resistance; iodide produces lysis of the granuloma; bismuth lies between arsphenamine and mercury. So, in general, the intensity of attack is favored by combinations of spirillicidal and resistance-building drugs.

Local vs. General Medication. In general, it may be said of all local antisyphilitic treatment that it is being slowly superseded as general treatment becomes more effective, and its only value lies in its ability to reach a few spots inaccessible because of deficient blood supply.

Collateral Factors in Treatment. Factors of great importance are the relation of strain and trauma, and of focal and intercurrent infection, etc., to the incidence and course of syphilis of the eye.

Arsphenamine in Ophthalmologic Practice. This drug, the author draws from his experience, is only contraindicated in primary atrophy. Definite conclusions as to the effect of arsphenamine on the eye cannot be drawn until examinations of vision, fundus and perimetric fields be done on a series of cases before and after arsphenamine. The original "606" is the most effective form of drug.

Effective Use of Mercury. This drug is best used as a daily injection of a soluble salt or as inunctions. The former is rapid in effect, while the latter is slow but powerful. Arsphenamine may be used simultaneously with a soluble mercurial.

Status of Bismuth. Present-day conclusions are that it is a better spirillicide than mercury, but definitely inferior to arsphenamine. Its chief advantage in ocular syphilis is as an excellent opening in acute processes not advancing too rapidly.

The Use of Iodide. Investigation shows that to produce penetration of the drug larger doses must be used, ranging from 15 grains t.i.d. even to 500 grains t.i.d. Intravenous administration has proved valuable in those phases of eye disease which are aspects of neurosyphilis.

Active Inflammatory Lesions; Optic Atrophy. Surprising degrees of recovery in secondary optic atrophy are often seen; but in primary optic atrophy the outlook is *nil* for recovery and only fair for arrest. The author opposes the use of arsphenamine at the outset in these cases, and has seen little advantage from intraspinal and intracranial therapy, extolled by some observers.

Treatment of Interstitial Keratitis. Especially here should ophthalmologists avail themselves of the modern methods of syphilotherapy. This condition can be reduced in extent and severity to a surprising degree by the simultaneous use of the aforementioned drugs. Involvement of the eye may apparently be prevented and the frequency of relapse much reduced.

Tryparsamide. This drug has no virtues in ophthalmic syphilis. Danger of injury to the optic nerve is proving to be less than expected and can be practically disregarded after the tenth injection. A coöperative patient is essential to the best protection of the eye in these cases.

The author trusts that the frankness with which he has discussed the viewpoint of the syphilologist in dealing with ocular syphilis has not impressed his hearers as merely bumptious or calculated to arouse antagonism or ill-will. If from an exchange of ideas some

element of the always priceless, because intelligent, reconciliation of general and special viewpoints can arise to the advantage of the victims of ocular syphilis, he will feel at once richly rewarded and generously forgiven.

Discussion. Dr. Kolmer stated that the specific treatment of early and late syphilis of the eye and optic nerves is essentially similar to the treatment of syphilis in general, except that greater caution is needed in the use of the pentavalent arsenicals like tryparsamide because of their more injurious effects upon the optic nerves than displayed by the trivalent compounds like arsphenamine and neoarsphenamine. He also pointed out the advisability of initiating the treatment of chronic syphilis of the eye by the administration of mercury and bismuth in association with one of the iodides in order to minimize or prevent the occurrence of neural Herxheimer reactions that may develop if the arsenicals are employed at the outset in large doses.

He believes that in the treatment of syphilis of the eye and optic nerves arsphenamine and neoarsphenamine are the arsenicals of choice, but that mercury and bismuth are likewise valuable medicaments. He is convinced that tryparsamide may produce toxic amblyopia and that it should be used only with great caution when the eye is involved. He also believed that it is advisable to have the eye examined before tryparsamide is administered in the treatment of other types of chronic syphilis.

In his experience the treatment of primary optic atrophy has been frequently disappointing, although the disease has been arrested in some cases, with improvement of vision, by intensive systemic medication with mercury, bismuth and neoarsphenamine in association with maximum doses of sodium iodide, the latter being sometimes given by intravenous injection. When this method fails to arrest the progress of the atrophic changes, he believes it is well to institute treatment by the Swift-Ellis method in addition to systemic medication.

FEBRUARY 17

Memoir of William M. Sweet, M.D.

By HOWARD F. HANSELL, M.D.

Dr. William M. Sweet was born June 24, 1860. He died December 24, 1926, of pneumonia after a week's illness.

It has been suggested by our chairman that we dedicate a few minutes of our time at this meeting to his memory.

The chairman has chosen me to represent you in recalling the presence and activities of our friend and distinguished colleague

because of my close association with him for more than a generation. The situation is difficult, if not painful, for in reviewing his life I become vividly conscious that our happy personal and professional experiences will never be repeated, and, just as in the case of the death of a dear relative, time must pass before memory will become blunted and the realization that he has passed and will not return. I am quite sure that my sentiments and feelings differ in no degree or kind from those of the other members of this Section who have been his friends for an equally long time.

It is fitting that we should halt every now and then from our engrossing professional work and direct our thoughts to the impermanency of life and consider how we can become wiser and more useful and more kindly to our fellow-creatures.

Dr. Sweet's deep interest and active participation in the purposes of this Section furnish us with an example and model that we would do well to imitate. He became a fellow of this College in 1898, and a member of the Section on Ophthalmology almost from its inception. For several years he was its efficient clerk, and then its chairman. Throughout this time, as is known to you as well as to me, he has read papers of great value and entered into discussions only when he felt he could add to their value. He is so well known to you that a history of his career and comment on his achievements in medicine seem unnecessary, yet it is becoming that we, friends of a lifetime, witnesses of his accomplishments and coworkers in his hospital and college activities, should officially do him honor who so highly honored the profession of medicine.

The choice of his calling was made while yet a young man and not much more than a boy, and the accomplishment of his purpose was beset with many—but not to him—unsurmountable obstacles. His schooldays were ended while still young and he was compelled by circumstances to help fill the family purse. By study and application he made himself familiar with the intricacies of the iron and steel trade, and during his student days contributed noteworthy articles to journals devoted to that subject and worthwhile editorials to *The Philadelphia Record*.

Handicapped financially, but with fixed purpose, he became a student in the Jefferson Medical College and graduated in 1886. He then served as intern in the Jefferson Hospital and completed the full term of two years. He lost no opportunity to learn from his distinguished chiefs, S. W. Gross, Bartholow, Da Costa and Brinton.

His power of observation was acute; his memory, cultivated assiduously, retentive; his ability to separate the chaff from the wheat, to recognize the worthy and ignore the unworthy, continued unabated throughout his professional life.

His early practice was meager, like the most of us, and during the lean year or so following the completion of his term as intern he supplemented his limited income by contributions of scientific articles to the trade journals. He became attached to the Eye Department of the Jefferson Hospital, and by his industry and ability rose from the humblest to the highest position—from clerk to assistant, then chief of clinic, associate, clinical professor, and finally—and alas! for only one and a half years—full professor.

His interests were not limited to the Jefferson. As one of the surgeons to the Wills Eye Hospital, his energy, operative skill, long experience and intelligent analysis of symptoms and signs of disease made him peculiarly valuable as a consultant. Also, after serving in various capacities in the eye clinic of the Polyclinic Hospital, he was made professor of diseases of the eye. The course of lectures, principally concerned with injuries of the eye, was highly instructive to the large audiences.

The meetings of the American Ophthalmological Society, of which he became a member in 1900, were a source of the greatest pleasure. He was regular in attendance, contributed many practical clinical papers, ably discussed the contributions of others, and enjoyed to the utmost the personal contact with his many friends in the Society. For ten years he served as secretary and treasurer, 1908 to 1918. For five years he was a member of the council. In 1920 he was elected vice-president and in 1921 president.

I gratefully insert a paragraph from a personal letter from our esteemed colleague, Dr. T. B. Holloway, who from intimate association with Sweet is well qualified to speak. He says: "I shall not go into anything relative to Sweet's fidelity, loyalty and faithfulness to every trust he carried for this Society. He labored many hours for its welfare; in fact, I know of no other member during my membership who has contributed so much to the welfare of the Society as Sweet."

As all the world of ophthalmology knows, Dr. Sweet's main professional interest was in the diagnosis of the presence, the situation and the extraction by magnet of foreign metallic substances in the eye. His localizer has been generally adopted and his magnet

almost universally used, at least in America. The former in his hands determined with the greatest accuracy the exact situation of a foreign body when it was dense enough to cast a shadow, and the latter, operating through a scleral incision and without insertion of the magnet tip into the vitreous, was sufficiently powerful to attract even the smallest fragments of iron or steel.

It seems superfluous for me to speak to you of Dr. Sweet's character. Nothing that I could say would add to your admiration of and esteem for him as an ethical, upright, industrious and genial colleague.

The esteem in which he was held by Joseph Meller, of Vienna, whose work on *Ocular Injuries*, as edited by Sweet, passed through two editions, is reflected in a letter just received, in which he says in part: "With Dr. William M. Sweet I have lost one of my best friends. I am deeply mourning for his decease and I shall never forget him. R. I. P."

I would like to close these brief and incomplete remarks with the beautiful expression of condolence, admiration and sympathy written by Anatole France to the widow on the death of Bjornson, the great Norwegian: "We feel that our hope for a better future is justified as long as there are men such as he who has upheld the cause of justice, and who was a firm believer in the advent of the day when labor will be extolled and poverty not to be a shame and when man will enjoy in peace the fruit of his good works."

Normal and Pathologic Anatomy of the Optic Canal in the Roentgenogram

Dr. Harry A. Goalwin, of New York, pointed out that the opening through which the optic nerve passes from the chiasm into the orbit, frequently referred to as the optic foramen, has, in most normal skulls, a definite length. It is, therefore, truly a canal. The shape of its cross-section is, in most normal skulls, that of a quadrant of a circle, the roof and mesial wall being nearly straight, while the floor and outer wall form the arc. This arc is the mesiosuperior side of the bridge which separates the optic canal from the sphenoidal fissure. The ophthalmic artery, which usually enters the orbit along the floor of the optic canal, has occasionally a separate canal, partial or complete. When this separate canal is incomplete, the two spurlike structures which form it should not be mistaken for

exostoses. The bridge which separates the optic canal from the sphenoidal fissure (later referred to simply as "the bridge") may also be incomplete, in which case the condition should not be confused with erosion of the bridge. Erosion of the bridge may be from within the optic canal (by an optic nerve tumor), in which case the canal is enlarged, circular or nearly circular, and smooth-walled; or from behind, below and temporal (by a pituitary tumor), in which case the tumor is demonstrated in the roentgenograms of the sella turcica.

The average size of the cross-section of the optic canal in normal skulls over three years old is 4.1 by 4.5 mm. Differences up to 25 per cent are found between the sizes of the two canals of the same individual. The average length of the normal optic canal is 4.3 mm.

In the normal optic canal the posterior margin of the roof lies in front of the posterior margin of the floor. This relation is reversed in certain types of deformed skull, notably oxycephaly, plagiocephaly (oblique skull) and acaphocephaly. In these deformed skulls the optic canals are also longer than in the normal. The most important change, however, noted in these deformities is the narrowing of one of the diameters of the optic canal and the deformed cross-section.

In order to obtain a useful roentgenogram of the optic canal it is necessary to arrange the tube, head and film in such a way that the central ray coincides with the axis of the optic canal and is perpendicular to the film. In normal adults the axis is directed downward and outward, making an angle of 38 degrees with the horizontal plane of the head and an angle of 38 degrees with the median vertical plane of the head. In the developmental stage (up to the age of seventeen years) this axis varies in its direction according to the age, the exact average for each age being shown in the curves which I have worked out and published elsewhere.[1] In the deformed skulls there is a change in the direction of this axis.

Pathologic changes of the optic canal may be changes in size, shape of cross-section, length, condition of the walls or condition of the lumen. The slides shown illustrate some of the typical pathologic changes. It will be noted that usually changes of several elements are noted: enlargement is associated with change in shape; hyperostosis or exostosis with change in size, etc.

A normal optic canal of typical shape and average cross-section was shown in the lantern slide.

[1] Zeitsch. f. Augenh., 1924.

The next was that of a normal three-year-old child, almost identical with the first in shape and size. Fracture of the bridge, periostitis, hyperostosis, exostosis, enlargement by optic nerve tumors, constriction by periostitis, constriction by multiple osteomata, destruction of bridge, destruction of the roof, constriction by old fracture of the roof, deformities of oxycephaly, plagiocephaly and scaphocephaly, circumscribed osteosclerosis of the sphenoid with associated elongation of the optic canal were among the other slides shown.

Particular attention was called to the slides which showed how a normal optic canal may appear to be constricted when the roentgenogram is improperly taken. The importance of calculating the actual size of the optic canal from its size on the roentgenogram cannot be overestimated. In fact, the roentgenogram is worthless for the determination of the shape and size of the canal unless the precautions indicated are taken, and the data necessary for the calculation are at hand (as shown in the formula on one of the slides:

$$X = d - \frac{b}{a}(d + f).$$

Of great interest was also the roentgenogram showing erosion of the walls of the optic canal by what was clinically and microscopically found to be a melanosarcoma of the choroid.

Discussion. Dr. E. P. Pendergrass thought Dr. Goalwin's work in this particular field of roentgenology was to be commended, but at the same time he thought that one should be very careful in making interpretations of findings because of the fact that the slightest distortion of the optic canal can show conditions that greatly simulate pathological conditions. He thought one of the features that should be greatly stressed in the interpretation of lesions of the optic canal is the comparison of the two sides. Demonstration of lesions in the optic canal should be of great value to ophthalmologists, but interpretations should only be made in the light of the most careful correlation of the clinical and roentgenological examinations. Dr. Goalwin himself has repeatedly experienced great difficulty in demonstrating deformities of the optic canal in his examinations, and Dr. Pendergrass thought the general roentgenologists should only interpret lesions in this region after the most painstaking examination. He thought that some of the conditions in which Dr. Goalwin demonstrated deformation in the optic canal could be demonstrated more easily and with a great deal more precision by other methods in general use.

Dr. F. F. Borzell was in accord with Dr. Pendergrass in congratulating Dr. Goalwin on his development of an exact technic for a specific part, such as the optic canal. He said that those of us who have not had the large experience that Dr. Goalwin has had must be conservative in our interpre-

tations, in order not to bring discredit upon an otherwise useful procedure. It would seem that at present interpretations must be deduced from comparison with the opposite foramen, provided we have no reason to suspect pathology on the opposite side. If both foraminæ appear to be abnormal, our interpretations should be doubly conservative.

Dr. T. B. Holloway extended his congratulations to Dr. Goalwin on his splendid contribution to the study of these frequently difficult cases. It was his belief that these studies were not as important in the cases with lesions of the middle fossa, where not infrequently the usual *x*-ray examination and associated clinical symptoms gave valuable information, and hence they might be regarded as accessory findings and secondary in importance to those cases where the lesions were about the apex of the orbit, such as fracture, accessory sinus disease, periostitis and so on, where they were essentially of primary importance.

Dr. Holloway believed that those cases of craniostenosis that came under the observation of the ophthalmologist usually presented signs that were almost unmistakable, namely, the optic atrophy, exophthalmos, divergent squint and occasionally nystagmus, which are almost invariably associated with facial asymmetry, deflected septum, high arching of the roof of the mouth and a train of other manifestations.

Aside from paying tribute to Dr. Goalwin, his main purpose in arising was to emphasize before the group of *x*-ray men present the importance of this character of study to the clinical type of cases that had been referred to, and he expressed the hope that this technic, although highly technical, would be made available for the ophthalmologists and others who were most apt to come in contact with such cases.

Essential Atrophy of the Iris

Dr. J. Milton Griscom read a paper in which he gave the details of a case showing essential atrophy of the iris of the right eye. There were two areas of complete atrophy of all the layers of the iris, and two smaller areas of partial atrophy. The intraocular tension was 35 mm. Hg. and the nerve-head showed marked cupping of the lower and temporal margins.

The slitlamp examination showed no evidence whatsoever of any inflammation of the iris. The fine pigmentation of the iris and migration of pigment, which have been noted in other iris atrophies and in glaucoma, were not present, there being less pigmentation than is usually seen at the age of the patient under observation. The one marked feature was the almost complete destruction of the endothelial cells lining the posterior surface of the cornea.

Discussion. Dr. deSchweinitz stated that although typical essential progressive atrophy of the iris, with hole formation in its tissue, is not a

common affection, its occurrence is evidently more frequent than the literature indicates, in that a number of examples of this variety of atrophy have been observed but have not been recorded. The left iris appears to be more frequently implicated than the right; rarely, it would seem, both sides are involved.

An essential attribute of this affection is "hole-formation." If there is only atrophy of the anterior layers of the iris the case does not belong to this group. It is definitely demonstrated that the iris atrophy antedates the establishment of glaucoma. Certainly in characteristic instances of this malady the atrophic process originates the glaucoma, and not the glaucoma the iris atrophy. As the atrophy proceeds, the root of the iris is welded to the periphery of the cornea, a dissemination of pigment occurs, the iris surface available for resorption of the intraocular fluid diminishes and glaucoma results.

The pigment grains which have been found in the posterior surface of the cornea were first noted by Harms, and are a product of the progressive atrophy and not due to a low-grade iritis.

Dr. Griscom has offered the first satisfactory study of the iris lesions by the slitlamp. In his own case, Dr. deSchweinitz said slitlamp investigations were not available, and when they were, the haze in the cornea had so far developed that an investigation of this character was useless.

In spite of Kreiker's interesting suggestion as to the etiology of this affection, quoted by the essayist, it would seem that the true cause of essential progressive atrophy of the iris and the formation of holes in its tissue terminating in glaucoma has not been discovered.

Dr. William Zentmayer stated that, in 1914, he showed before the Section a case of essential atrophy of the iris which in history and iris involvement so closely resembled the case reported by Dr. deSchweinitz a few years later that for a time they thought that they might have seen the same patient until Dr. deSchweinitz noticed that opposite segments of the iris were affected.

In the possible causes enumerated by Dr. Griscom he mentioned vascular disturbance, for which Dr. Zentmayer believed he was responsible, having suggested it as a possibility in his own case. The case, however, was not thoroughly studied from the physical standpoint, and, as Dr. deSchweinitz stated in his paper, Dr. Zentmayer failed to assign any cause for the vascular condition.

Dr. Luther C. Peter put on record an unpublished case similar to that reported by Dr. Griscom, which was under observation in his service at the Polyclinic Hospital.

The reaction of this patient's eye to surgery was very interesting. It was a painful eye, and very hard and red. An effort was made to temporarily reduce the tension by a paracentesis of the anterior chamber. Immediately after the withdrawal of the needle the blood oozed from all parts of the iris, about half-filling the anterior chamber. A second attempt was made to reduce the tension by a posterior sclerotomy. This held for a while, but the increased tension again returned. The case will be formally placed upon record after the studies have been completed.

Ring Scotoma in Glaucoma. Report of a Case

Dr. H. Maxwell Langdon stated that ring scotoma was first reported by von Graefe in 1858, occurring in 2 cases of pigmented retinal degeneration. Since then it has been reported in many other conditions—optic nerve lesions, retinochoroiditis, idiopathic night-blindness, migraine after lightning flash, disease of the sphenoidal sinus, after eclipse of sun and glaucoma.

Attention was called to it as occurring in the last-named condition by Bjerrum in 1890, at the International Congress of Ophthalmology, in his paper on the "Visual Field." He described a scotoma always connected with the blind spot, arcuate in form, curving above and below the fixing part, or, as occurred in some cases, one such above and another below, forming a complete ring. The condition he believed to be one due to pressure on some of the tracts of nerves as they curved over the lips of the cup.

Colonel Elliott agrees with this explanation, but thinks that any pathological condition interfering with definite nerve bundles on or near the disk may leave a similar result, and mentions retrobulbar neuritis, hemorrhagic neuroretinitis and retinochoroiditis juxta papillaris as examples of this.

Bjerrum has also reported it in a case of embolism of the central artery.

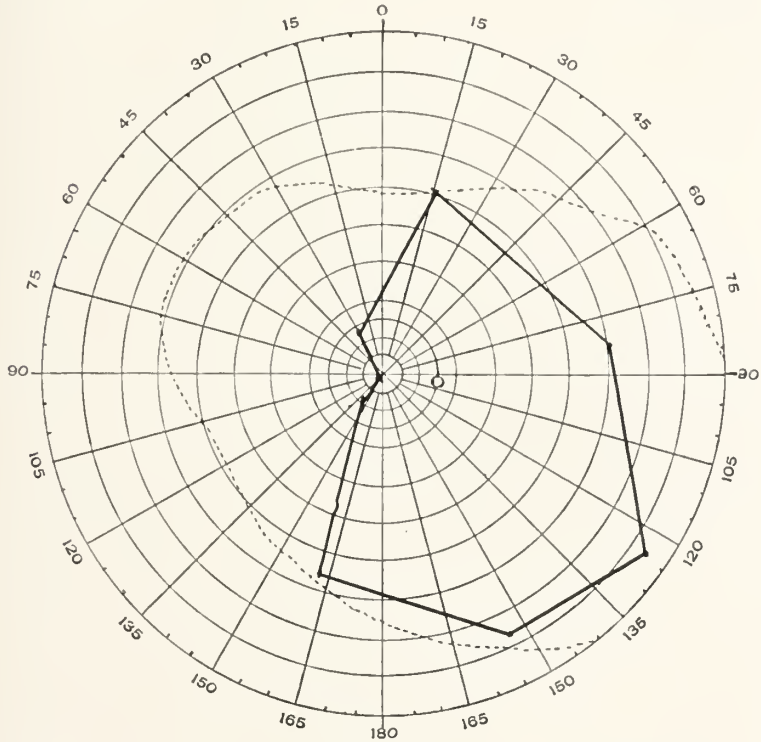
It is not a common manifestation in glaucoma, cases having been reported by Noges, Mittendorf, Handman, Zentmayer, and Posey, Pfluger, Bjerrum and deSchweinitz.

The case here reported is that of Mr. H. W., who was first seen January 15, 1921, his age at that time being seventy-two. His complaint was discovered to be partially the result of some uncorrected hyperopia and partially of a very advanced arteriosclerosis, the retinal vessels being markedly involved. He was seen at intervals until March, 1923, when, on account of continued headache, he sought other ophthalmic advice, but returned to the writer in August, 1925. During this interval, among other therapeutic measures, leeches had been applied to the temples at frequent intervals. The condition which caused his return was a blurring of vision, especially of O.D. which was found on examination to be 5/30, that of O.S. 5/15; changes in the correction improving this to 5/5 and 5/8 respectively. There had been a hemorrhage into the right vitreous and macular changes in O.S. Both improved,

sodium iodide being the therapeutic agent, O.D. vitreous becoming quite clear and the punctate hemorrhages of the left macular region absorbing until vision became 5/6+ and still is so.

On October 16, he complained of heaviness and pressure around O.D. and examination showed a cut in the nasal field and a tension of 85, the tension of O.S. being 30, and O.S. field full; the pupil of

No. 1 Form, X–20–2



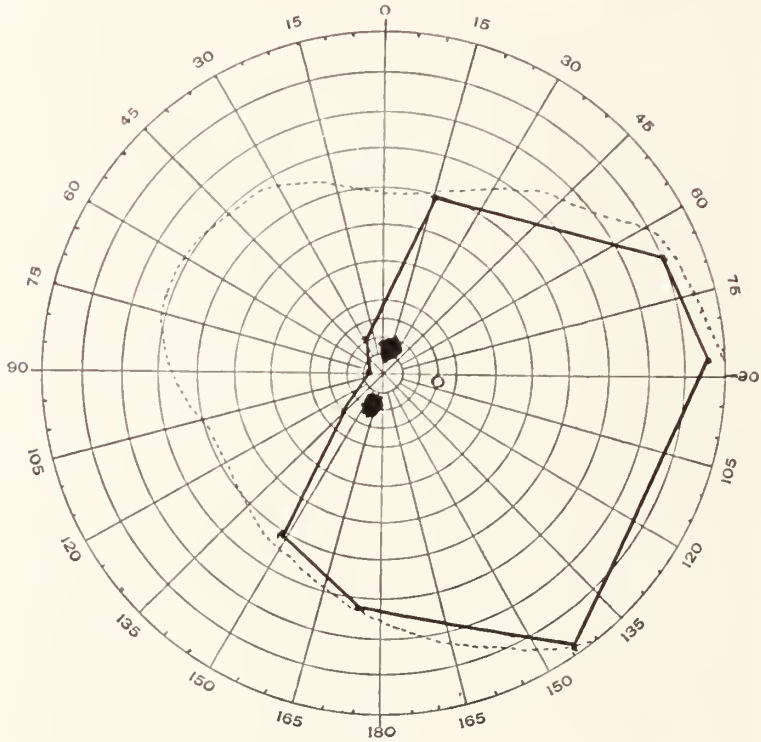
RIGHT CHART 1.

O.D. not being over 3.5 mm. and there being no other objective signs of glaucoma. Pilocarpine, 2 grains to the ounce, was prescribed, 1 drop four times daily, which reduced the tension of O.D. to 40 and O.S. to 23 in forty-eight hours. Two scotomata had appeared in the field of O.D., as shown on Chart No. 2, but central vision was still 5/5. The myotic, pilocarpine, was changed to eserine, 1 grain to the ounce, and operation was suggested, the

patient being asked to see Dr. deSchweinitz in consultation, for while a trephine was felt to be the safest operative measure, even this was regarded as risky considering the previous hemorrhages and the bad retinal vessels.

Dr. deSchweinitz found conditions as above described, including the partial Bjerrum scotomata. He felt that trephine was the opera-

No. 2 Form, X-26-25



RIGHT. CHART 2.

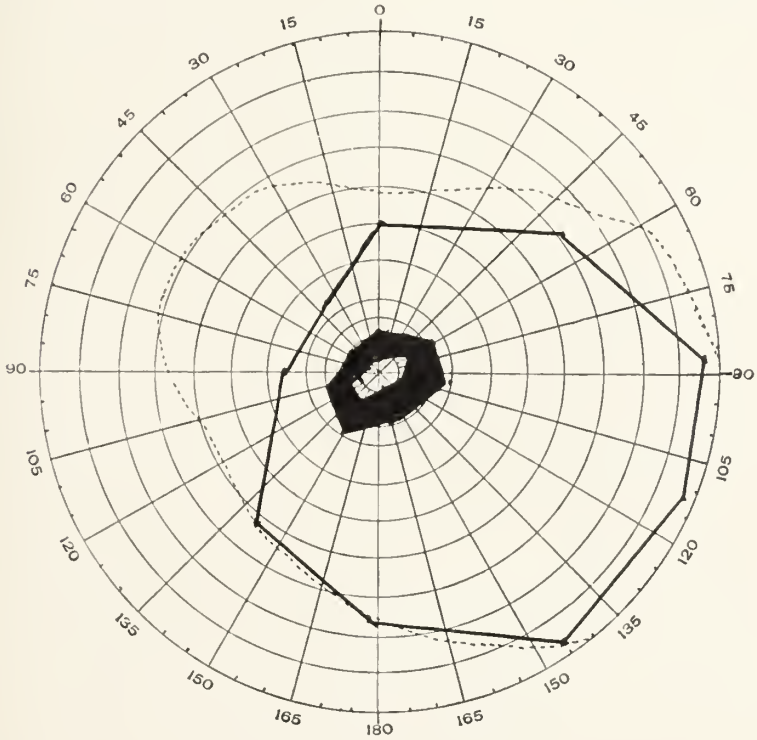
tion of choice but advised continuance of the myotic treatment and frankness with the patient concerning the operative risk. Operation was, and still is, refused by the patient.

Under myotics the tension fell to 28 in O.D., and 20 in O.S. On November 19, rose to 43 in O.D. and a ring scotoma, as shown in Chart 3, developed; central vision remaining 5/5. The eserine was increased slightly and tension fell to 30, in which neighborhood it

has since remained, varying between 25 and 35. Vision has gradually failed in O.D. until it is now about 1/60, but operation will not be permitted, as the eye is comfortable and the left is in good condition; tension, 18; vision, 5/5, and field normal.

The most interesting thing, besides the formation of the scotoma, seems to be the complete absence of pain and the other complications

No. 3 Form, X-19-25



RIGHT CHART 3.

which are usual with such high tension as existed during the first attack.

It is rather difficult to explain the site of the first part of the Bjerrum scotoma to develop, it being both above and below, about equidistant between the blind spot and the point immediately opposite to it, or almost on the vertical meridian. It would seem that pressure on the bundles of nerves supplying these areas would

be more likely to affect the region immediately in the neighborhood of the blind spot, or else the whole arcuate area at once; just why the midportion first suffered, seems puzzling.

Discussion. Dr. G. E. deSchweinitz expressed the opinion that ring scotoma in glaucoma was not a very uncommon visual field defect. He exhibited a number of field charts which indicated that such annular scotomas were formed by the union of inferior and superior Bjerrum scotomas. He showed one visual field chart, the patient being the subject of chronic glaucoma, which portrayed a wide zonular scotoma midway between fixation and the periphery, unconnected with the blind spot. It was possible, he thought, that the scotoma in this case was of retinal origin.

Dr. William Zentmayer recalled the case of J. H., a man seventy years of age, with tension in each eye of 37 mm. Hg. Central vision; O.D., 5/15; O.S., 5/6. In each eye there was a deep glaucomatous cup and marked angiosclerosis of the retina.

The fields taken on the perimeter showed the upper nasal field of the right eye almost entirely gone, and also a peripheral contraction, with no color perception. There was a large paracentral scotoma of both eyes, absolute in the right and relative in the left. The left eye showed, in the peripheral field, a concentric contraction for form and color.

One year later there was in the left eye a ring scotoma of an average width of about 12 degrees, two arcs meeting in a point 15 degrees to the nasal side of fixation.

Two and a half years after this the red field in the left eye was reduced to a hemianopsic area 8 degrees from fixation on the temporal side, and the form field had also become almost hemianopsic. The blind spot was increased about 200 per cent.

At the time of his last visit, in April, 1924, vision was O.D. finger counting, and O.S. 5/6. Tension was, O.D., 51; O.S., 45.

Dr. Luther C. Peter, in answer to Dr. Langdon's question as to the reasons for Bjerrum scotomas appearing above and below the blind spot instead of adjoining the blind spot, pointed out that Dr. Elliot very clearly explains the reasons for the possibility of the Bjerrum scotoma appearing in any part of the field. This same fact was emphasized by Dr. Peter two months ago in a paper before this Section.

The bizarre types of Bjerrum scotoma, as well as the usual forms, depend entirely upon the position and the direction of the development of the cup, due to the strength or weakness of the various zones of the cribriform membrane. The direction of the entrance of the optic nerve into the sclera is a second factor. The scotoma, therefore, may select any bundle of nerve fibers, and the mid-upper and mid-lower temporal quadrants are the bundles which are most frequently affected. In his experience, as stated on previous occasions, the usual Bjerrum scotoma is just as apt to evolve from a point above or below the point of fixation in the field between the tenth and the twentieth degrees, as well as from a point adjoining the blind spot.

Linear Precipitates on the Posterior Surface of the Cornea

Dr. Luther C. Peter presented notes on a case of bilateral pigmented precipitates on the posterior surface of the cornea in a woman, fifty-four years of age, a hyperope. The patient's condition is one of right-sided hemiplegia as a result of advanced cardiovascular-renal disease. The precipitates on the posterior surface of the cornea are common in a pyramidal zone, especially occupying the lower half of the cornea. Precipitates, however, fusiform in distribution and occupying the center of the pupillary spaces, are unusual.

Vogt, Kruckenberg and others have observed these precipitates. Some regard them as congenital in origin, others as acquired. Vogt believes that the precipitates occur only in old age, in high myopes, bilateral in distribution and usually fusiform. He believes them to be acquired.

In the patient presented, fifty-four years of age can hardly be called senile, and the patient is a hyperope. The precipitates can be recognized by a 20 in the ophthalmoscope as a dark-brown, sharply-defined line, occupying the vertical meridian of the cornea. With the slitlamp this line is observed as fusiform. The precipitates are red, stellate in character, in all probability washed out and scattered over the surface by the aqueous, because the lines do not extend to the limbus from above or below.

The reason for the linear distribution, in all probability, is to be found in the aqueous circulation. One usually observes this circulation more active in the midzone than in the lateral zones. This is due to the fact that the circulation is more easily recognized in the dark pupillary space; and second, because the entire corneal surface is more remote from the iris, and the temperature probably is considerably lower than in the areas near the limbus.

A Case of Unusual Distribution of Opaque Nerve Fibers of the Retina

Dr. H. Maxwell Langdon exhibited a patient, Miss M. R., aged twenty-three years, who was seen on December 10, 1926, complaining of some headaches and some blurring of vision of the right eye. Vision of the right eye was 5/15, corrected to 5/6+. The vision of the left eye was 5/5. There were no external changes.

The media were clear in each eye and the right disk was surrounded by large masses of medullated nerve fibers extending about a disk diameter from the disk above, with winglike projections on each side between three and four times this amount.

There were no other fundus changes in the right eye, and the left fundus was normal. In spite of the subnormal vision, no scotoma covering the part of the field corresponding to the affected portion of the retina, could be demonstrated, though this was tried with different illuminations and very small test-objects. The only interest in the case is the very wide extent of the involved area.

Case of Coloboma of the Iris, with Equatorial Punctate Cataract

Dr. Sidney L. Olsho stated that colobomata of the iris are among the most frequent congenital ocular defects. This Section has seen many of them. Cases have been presented by Drs. Oliver, Posey, deSchweinitz and others. Each additional case, however, has a new fascination that stimulates our curiosity, for their etiology is still speculative and the theories that have been advanced to explain them are only slightly less numerous than the authors presenting the cases.

The fact that most colobomata are situated below gives rise to the theory of incomplete closure of the cleft of the optical vesicle, but their occurrence at other sites seems to dispose of this theory.

The one mentioned by deSchweinitz is that of Lang and Treacher Collins, which is that there is either an abnormal adhesion between, or late separation of, the lens and cornea, the iris failing to develop in the area involved.

Then there is the one to which Friedenwald gives preference, namely, that there is a possible persistence of the vascular connections which exist in the early embryo between the stroma of the iris and the hyaloid system on the back of the lens, and that, accordingly, the persistence of these vessels inhibits the forward growth of the edge of the optical vesicle, bringing about an indentation.

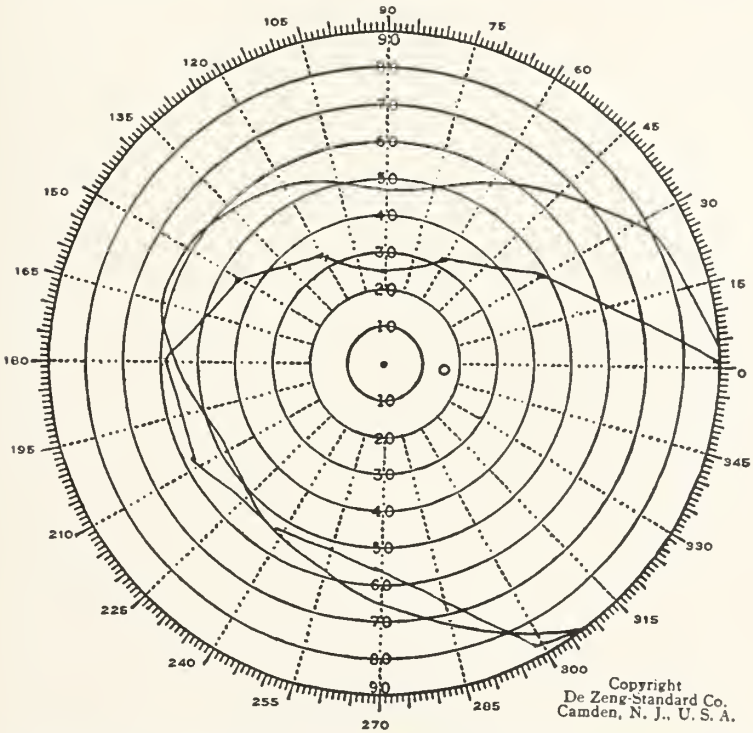
Posey states that in embryonal life the long and short ciliary arteries normally anastomose to form the circulus major, and that, from these, further twigs are give off to complete, with their surrounding connective-tissue cells, the iris stroma. If there be an

absence or anomalous distribution of these vessels, a coloboma develops at the corresponding site.

He did not enter into a discussion of these theories, but wished simply to present this classical case with attention drawn to features that he thought unusual.

No. 3450
Name, A. E.
Address
2-14-1927

Size Disk used, 10 mm.
Illumination, 200 W. Elec.



Right.—Dr. Olsho's modified chart for use with McHardy's registering perimeter. (Published by Wall and Ochs, Opticians, 1716 Chestnut Street, Philadelphia.)

Chart 4.

The patient, Miss A. E., aged sixty-two years, was first seen by him in the eye department of Jefferson Hospital, Dr. Howard F. Hansell's service, to whom he was indebted for this privilege.

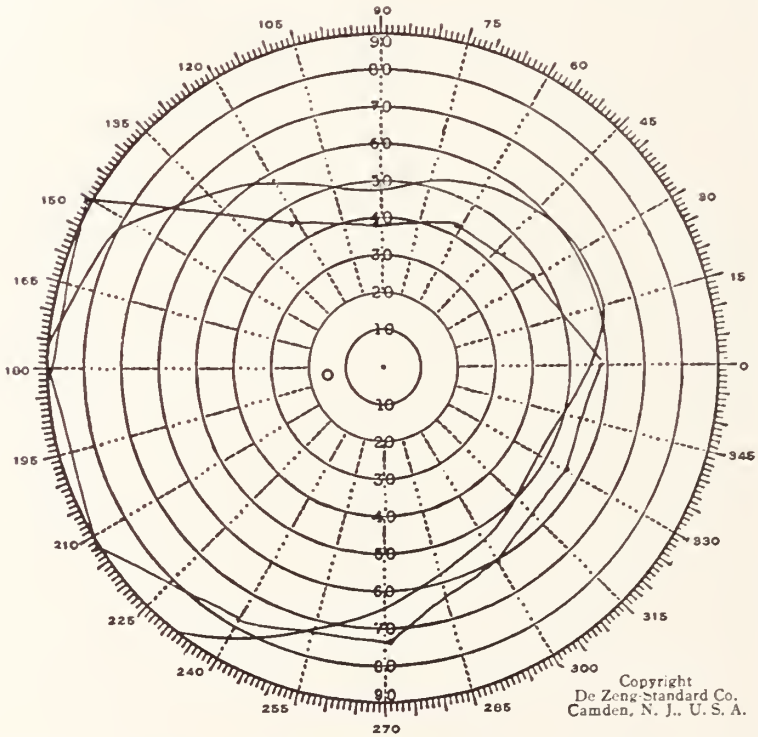
She came for a new refraction, stating that her left eye had been uncomfortable with the glasses previously prescribed.

The right eye appeared to be normal. The pupil measured 4 mm. in moderate illumination and responded to the usual stimuli.

The left eye presented the keyhole pupil characteristic of the condition. There was a coloboma limited to the iris, below and slightly inward. The width of the coloboma at the pupillary border

No. 3450.
Name, A. E.
Address
2-14-1927

Size Disk used, 10 mm.
Illumination, 200 W. Elec.



LEFT.—Dr. Olsho's modified chart for use with McHardy's registering perimeter. Degrees correspond to trial frame. (Published by Wall and Ochs, Opticians, 1716 Chestnut Street, Philadelphia.)

CHART 5.

was 3 mm., at the ciliary border 2 mm. The retinal pigment was continued from the pupillary arc along the margins of the pillars of the coloboma as far as the root of the iris. The central portion of the pupil reacted to the various stimuli. The pupils reacted consensually.

The width of the iris above the upper margin of the right pupil was 3 mm., while the width of the iris above the upper margin of the left pupil was 4 mm. Vision was as follows: R.E. 20/20 corrected by 0.25 c. ax. 180 = 20/20. L.E. 20/70 corrected by 1.00 sph. 0.50 c. ax. 15 = 20/20.

The form fields presented herewith show, as expected, a fulness of the left field below. In the vertical meridian it extended downward 20 degrees farther than in the same meridian for the right eye. The ophthalmoscope shows

Right Eye: Media clear. The fundus was normal. One small cilioretinal artery was present at the temporal edge of the disk.

Left Eye: The fundus was normal. There was some intertwining of vessels and a deep physiological cup. The central portion of the pupil presented clear media. The equatorial portion of the lens, starting at the upper limit of the coloboma, showed a moderately wide band of diffuse opacity, then a narrow band more clear, and then again a narrow band of increased density. The dotlike character of the central band could be made out ophthalmoscopically.

The case presents an unusual opportunity for slitlamp study of the periphery of the lens. Oc. 2. Objs. 4 and 6.

In the equatorial region below, where the anterior and posterior bands of the peripheral embryonic nucleus join, there was a shower of yellowish-white rodlike and irregularly cuneiform opacities, their general arrangement suggesting a flying wedge with apex downward. Just below this wedge, in the adjacent zone of discontinuity, the opacities were less numerous; then, farther down, where the anterior and posterior bands of the senile nucleus join, there was another less heavy shower of opacities, also suggesting a flying wedge.

The slitlamp study seemed to verify the ophthalmoscopic appearance of a clearer zone between two denser zones. Throughout their extent the senile nuclear bands, both anterior and posterior, presented a few punctate opacities and a slight increase in density. Study of the right lens showed a general increase of density of the anterior and posterior senile-nucleus bands.

MARCH 17

Angioid Streaks of the Retina

Dr. J. Milton Griscom presented the details of a case of angioid streaks of the retina in a colored man, aged sixty-eight years, who

had been exhibited before the Section at the March meeting in 1926. The color and distribution of the pigmented lines in the retina conform generally to those which have been previously described by other observers.

In contrast to most of the other cases reported, there was no evidence in either eye of past or present retinal hemorrhages or of any choroidal disease. The patient exhibited an irregular, slate-colored pigmentation on the skin of the forehead, which had been diagnosed as probable chloasma. The explanation as to the formation of these streaks, as given by various writers, was discussed. Dr. Griscom suggested a possible common cause for the pigment proliferation on the skin and the pigmented streaks in the retina.

Discussion. Dr. William Zentmayer said he thought the regression changes noted in this case suggest a hemorrhagic origin for the pigment.

Dr. G. E. deSchweinitz, after a brief reference to various types of striæ in the retina, including angioid streaks, and some remarks on early publications which relate to the latter affection, exhibited three water-colors which portrayed in typical manner the lesions of this retinal manifestation, and which had served to illustrate an article published in 1895. The patients were brothers.

This observation and others of similar character in the literature indicate, he said, that angioid streaks in some instances may be regarded as a familial affection.

None of the explanations of the origin of these streaks, as Lederer, Fuchs and other observers have stated, are entirely satisfactory, and, although the streaks are probably essentially due to hemorrhages variously disposed, it appears to be definitely true that there are several different types of this disorder. For instance, in one type the streaks ultimately disappear, sometimes after long periods of time, as Dr. Zentmayer had demonstrated, and sometimes in a comparatively short period—two years, as in a case reported by Ascher. Again, gross macular changes—hemorrhages and exudations—may be an early and associated manifestation, or, as Coppez and Danis have noted, exudative macular retinitis may constitute a terminal stage.

Dr. deSchweinitz expressed the opinion that a careful analysis of all reported cases, whereby the various types and their relation to other retinal disorders could be established, as well as the evolution, course and termination of the lesions of this affection, would constitute a valuable research.

Angioid Streaks in the Retina

Dr. William Zentmayer showed a case of angioid streaks which he first showed at the Section eighteen years ago. He had been shown on two other occasions.

In the right eye the pigment zone about the disk had almost completely disappeared. One stripe, fairly characteristic but very pale, follows the course of the superior temporal vessel. Elsewhere in the fundus are seen short streaks, sections of former streaks, one gray in color, one terminating in a white band. At the fovea are oblong dense black pigment masses about 1 dd. in size.

In the left eye the pigment masses at the macula are larger and denser than in the right eye. There is one brownish streak running for some distance from the upper margin of the disk. A second short section of a streak is present near the equator.

In both eyes there is sclerosis of the choroidal vessels. Both disks are yellowish and the periphery of the fundus shows the yellow and black flecking originally seen.

No recent changes are noted in either eye.

Congenital Syphilitic Chorioretinitis

Dr. Jonas S. Friedenwald, of Baltimore, Md., reported the result of the histological examination of the eyes of 91 infants either stillborn or dying shortly after birth. Among these, 17 infants were found to be syphilitic, and 3 of the 17 showed choroidal and retinal changes attributable to the infection. The lesions were of several types: (1) small circumscribed chorioretinal scars; (2) diffuse infiltration of the choroid of myeloid cells; (3) diffuse interstitial scarring of the choroid (this was regarded as a late stage of the same process as presented in 2); (4) diffuse neuroretinitis. The relation of these pathological findings to the familiar ophthalmoscopic picture of congenital syphilitic chorioretinitis was discussed, as was also the significance of the myeloid cells in the chorioretinal lesions.

Discussion. Dr. Francis Heed Adler asked Dr. Friedenwald whether he had noticed any tendency of the lesions in syphilitic fetal eyes to be grouped in the anterior segment of the globe. Igersheimer has pointed out that the characteristic lesions of ocular syphilis are an interstitial keratitis and an anterior choroiditis. Igersheimer was not able to explain why the lesions were nearly always in the anterior segment of the globe, but it would appear from Dr. Friedenwald's slides that the posterior segment could also be involved in fetal life.

Digest of Some Clinical and Experimental Observations on the Use of Foreign Protein, Especially in Iritis

Dr. Hunter W. Scarlett briefly reviewed the literature, showing that the use of foreign protein is by no means new, dating as far back as 1904, when Darier started his experiments with diphtheria antitoxin. Experimentally no definite conclusion as to the value of foreign protein in ocular infections in animals was arrived at which could compare with the success which had been attained in treating humans.

Thirteen cases in all were treated, 10 of which were iritis; 3 of the latter were complicated by foci of infection and, although they responded favorably to foreign-protein injections, did not entirely recover until every focus had been thoroughly sought and eradicated. The remaining 7 cases of iritis, all of which were severe, recovered quickly, cutting down the convalescing period to an average of fourteen days.

The most striking feature in all the cases treated with foreign protein, especially those of iritis, was the rapid relief from pain. One patient said the pain disappeared within one hour.

The conclusions were: (1) That foreign protein was a valuable adjunct in treating ocular infections, but was not a "cure-all," and should never be used to the exclusion of the routine methods; (2) in our cases, especially those of iritis, we felt that the convalescing period was materially shortened and the severity of the symptoms markedly lessened; (3) of the foreign proteins used, horse serum gave the most marked reaction and also the quickest relief from symptoms; (4) all foci of infection should be removed; (5) in our experiments on rabbits' eyes no benefit from the use of foreign protein was detected.

Discussion. Dr. Edward A. Shumway asked Dr. Scarlett in what doses he had used the injections of aolan. He had tried it in several instances and thought he had got very good effect, especially in a case of bad iridocyclitis following cataract extraction. In this case he had first used an injection of horse serum, and, on giving 10 cc. of the aolan, very sharp reactions were produced, with rise in temperature and general disturbance.

Dr. Scarlett, in answering Dr. Shumway's question about dosage, stated that 5-cc. doses were used intramuscularly.

Trachoma Among the Indians of the Southwest

Dr. W. C. Posey gave a short account of a trip which Dr. Proctor, of Boston, and he, as part of a committee appointed by the American Medical Association to confer with the Department of the Interior, had made to the Southwest to study the spread of trachoma among the Indians. An inspection of the chief schools, hospitals and villages in that region showed that Indians everywhere, both adults and children, were affected, all stages of trachoma being observed. The physicians of the Indian Service, with whom they came in contact, seemed familiar with the disease and no cases of mistaken diagnosis were observed. For the most part the disease was treated conservatively and well. Dr. Posey thought that, notwithstanding the excellent sanitary conditions of the schools which were visited, perhaps the main cause of the more rapid spread of the disease in recent years has been the failure to separate children with trachoma from those unaffected with the disease in the schools, and he commended very highly the recent order of the Government to segregate all trachomatous children hereafter in separate schools. The establishment of a trachomatous hospital school for this purpose at Fort Defiance, Arizona, initiates this movement. Pupils will be kept under close surveillance here, treated for trachoma, and receive as much educational and vocational training as their ocular condition warrants. Other institutions of a similar nature will probably be established throughout the Indian region.

Dr. Posey spoke of the conditions attending the treatment of adult Indians; their life upon the plains, where they are exposed to the severity of the weather, exacerbates the disease. Their belief in the spiritual as well as in the healing powers of their "medicine men" often cause them to neglect medical advice. Their cabins are usually unsanitary. Until living conditions are improved, trachoma will surely thrive.

Dr. Posey deprecated so-called "clean-up" campaigns, insisting that after-treatment is just as essential as primary measures. Tarsectomy should be reserved for the later stages of the disease, the earlier being treated by expression, mild grattage and by astringents —never by radical surgical procedures. A series of hospitals should be established for the treatment of eye diseases, which should be easy of access, with ample provision for indoor treatment. Efforts should be made to teach adult Indians proper methods of living by

the establishment of community centers where they may be taught, by those who speak their own language, methods of personal hygiene. Graduates of the Indian schools could be utilized for this purpose. Not until the Indians have adopted the standards of the whites will trachoma be eradicated.

APRIL 21

Congenital Albinism

Dr. Walter J. Sener presented an infant, W. B., aged four months, who was referred to him by a local physician for a diagnosis, as he was particularly interested in the color of the eyes and the nystagmus which the child displayed.

As soon as Dr. Miller saw the child he made a diagnosis of complete congenital albinism, and so advised the doctor. He then advised the mother to put dark glasses on the child and to keep it protected from the light as much as possible, by making a bonnet for it.

The child is the only member of the family that has this condition on either the mother's or father's side of the house, as far back as could be traced.

The condition is well known to the older ophthalmologists, and Dr. Miller presented the case for the benefit of the younger men and the attending students.

Cataracta Cœrulea

Dr. Walter J. Sener exhibited Miss E. H., aged twenty-two years, who was sent to him by a local optician who tried to improve her vision but found it impossible. He found it to be a case of cataracta cærulea, or blue cataract, and took her into the Polyclinic Hospital in order to study the case thoroughly, for he noticed that she had a choked disk as well. Every possible laboratory test was made to try to determine the cause of her condition, but the only thing found was that she had decidedly concentrically contracted visual fields contracted down to about 8 degrees on a Peter hand campimeter, a few hyaline casts and a trace of albumin in the urine.

She was kept in the hospital for about ten days or two weeks and then refracted, giving her vision of 20/20 in O.D. and 20/40 in O.S.

She was advised to go home and be available for study at any time, telling her family the exact condition of the cataracts.

She improved physically, but her vision, to the time of this report, remains the same.

Discussion. Dr. G. E. deSchweinitz referred to the frequency with which various forms of cataracta punctata are revealed by examination of the lens with the aid of a Zeiss loup, and especially by means of a slitlamp. He quoted Paul Roemer's statement that, with the exception of subcapsular cataract, it is the form of cataract most frequently seen in old people.

Referring to those varieties in which the roundish and point-like lesions have a bluish, often sky-blue, color—hence the term "*cataracta punctata cærulea*"—he described the various dispositions the lesions assume, namely, those that are seen chiefly in the anterior portion of the lens, the opacities being ellipsoid in form and arranged, to use Roemer's term, like "palisades;" situated in the preëquatorial zone, the equator being unaffected, between which are numerous collections of fine bluish points; to the various globular opacities and clouds of minute bluish dots, like star-dust; to the lesions often just beneath the capsule, but also more deeply placed, which resemble fragments of a fern leaf; to the formation centrally of a Y-shaped figure built by a coalescence of roundish lesions; and to the combination of coronal cataract (Vogt's cataract), being a wreath-like zone in the form of roundish or oblong opacities in the area of the "nuclear equator," and the lesions of cataracta cerulea—hence *cataracta punctata cerulea et cataracta coronaria*. All these manifestations, he said, constituted the most beautiful of slit-lamp pictures.

Although punctate cataract is often classified among the congenital forms of cataract, it is more probable that, while some varieties may be congenital in origin, the majority of them appear between the tenth and twentieth years of life, especially, as Butler has pointed out, because the opacities are largely in a portion of the lens unformed in infancy.

Usually the vision of patients with punctate cerulean cataract, even when the lesions are extensive, is good, and the development of the opacities is only slowly progressive, and Dr. deSchweinitz referred to several patients under observation for many years, in whose lenses the process had remained stationary.

He had been impressed by the slight and often casual reference to this form of cataract in textbooks, with a few exceptions. This type of cataract in its true characteristics is only satisfactorily detected if the lens tissue is studied by means of a loup or with a slitlamp. Judging from personal experience, the association of punctate cerulean cataract and high refractive error is not uncommon. In six patients recently studied, two had conical cornea, one a high mixed astigmatism and one a considerable myopia and astigmatism.

Dr. T. B. Holloway stated that he had described the various types of cerulean cataract to which Dr. deSchweinitz had just referred. Some years ago the literature on this subject was scanty and doubtless many cases

were overlooked. In addition to the contribution of Pearce, Rankin and Ormond, van der Scheer has reported concerning 60 cases observed in Mongolian idiots, and found that there was an increasing tendency among these cases to this type of lens change until after the age of seventeen, when practically all of them showed defects in the lens.

With the ophthalmoscope they are invariably missed, and the opacities appear much smaller than when seen by oblique illumination, and by this latter method the blue color when present can be readily recognized. Dr. Holloway felt that much was missed in the study of cataract cases by not using the oblique method of illumination.

He contrasted the diagnosis of cerulean cataract, where the diagnosis was dependent upon oblique illumination, with the Vossius ring cataract, where the diagnosis could but rarely be made by oblique illumination, this being determined by means of the ophthalmoscope.

In regard to the cause of the color, he was under the impression that Lord Rayleigh had attributed this to a difference in the refractive index of the fine particles causing dispersion of the short wave lengths.

Superficial Linear Keratitis

Dr. J. C. Knipe exhibited a case of superficial linear keratitis in a man, aged fifty-eight years, in which eight or ten vertical lines extended above and below a central area of the cornea, which stained in a punctate manner with fluorescein. The lines did not stain except about the central area. The lines corresponded to the description of the cases described by Spicer and Greeves. They were slightly raised, double contoured and tapering toward the extremities near to the limbus. The slight pain and injection, both conjunctival and ciliary, subsided in a few days, but the striations are still visible, although less prominent, three weeks later. The tension is slightly reduced.

The etiology could not be determined, unless many carious teeth were the disturbing factor. The patient was in good health. The blood and urine reports were negative. There was no history of injury.

Discussion. Dr. G. E. deSchweinitz stated that although folds and rupture of Bowman's membrane, following injury and in association with corneal scars, and sometimes with analogous folds in Descemet's membrane, appearing as double contoured lines, are not uncommon, the affection so well described by the essayist, and particularly investigated by Spicer and Greeves, with wrinkling of Bowman's membrane and the formation of new fibrous tissue in the adjacent substantia propria, is certainly unusual.

In his own experience only one case certainly of this character had

occurred, and followed injury. But although injury may be a contributing factor, as has been pointed out, it is difficult to believe it is the essential cause, which, indeed, is not assignable. In his own case the results of treatment, so long as the patient remained under observation, were of indifferent value. Evidently the letter or alphabet keratitis of Haab should be regarded as a process similar to, if not exactly like, the linear keratitis of Spicer and Greeves.

Exhibition of a Case of Neurofibromatosis

Dr. William Zentmayer exhibited a case which was referred to the Wills Hospital by Dr. Fordyce for the correction of ptosis. Examination showed the ptosis to be due to a hypertrophy of the outer half of the lid. On palpation, this was baggy and had a soft feel. The external orbital margin was thickened. Just behind the external angle there was a depression in the temporal bone. In the parietal region there was a slightly elevated hemispherical mass about 7 cm. in diameter composed of a plexus of dense, cordlike fibers with beadlike swellings along their course. The fundus was normal.

The report from the neurological clinic was negative. The *x*-ray showed complete erosion of the clinoid process.

Neurofibromatosis with ocular involvement is a rare condition. According to Fuchs, there is sometimes associated a lymphangioma of the lid, and Collins speaks of a general connective-tissue hypertrophy of the lid. It is well-known that neurofibromatosis is sometimes associated with buphthalmos.

While this condition of the eyeball is not due to elephantiasis, yet sometimes the ciliary nerves show fibromatosis.

The pathology is one of hypertrophy of the epi- and perineurium without involvement of the axis cylinder.

Discussion. Dr. Burton Chance desired to put on record 2 cases resembling Dr. Zentmayer's; the children, sisters, were younger than Dr. Zentmayer's patient and were brought to the Wills Hospital from the country by the district nurse, in 1920. The symptoms in each were confined to one side, and were quite the same. The upper lid drooped and had the wormy feel; there was hydrophthalmos, and the bone deficiency like that observed in the orbital region of the child here present. It is not recalled that there were anomalies in the scalp. Operations were suggested, but the parents of the children were afraid that terrible things were to be done to the little ones, so they withdrew them and have not been heard from since.

Dr. T. B. Holloway stated that the appearance of this case bore such a striking resemblance to a case he has had under observation for four or five years that the diagnosis was suggested as soon as the patient was seen. At the time his patient came under observation the lesion appeared at

the outer end of the right upper lid near the orbital margin, and naturally the possibilities of a dermoid cyst were suggested. In the careful dissection, needlelike filaments were dissected out, some of these attaining a length of 3 or 4 inches, but when *in situ* they were tightly coiled. The specimen was submitted to several pathologists before the diagnosis was subsequently made.

Since the first operation, a mass has been removed from the margin of the right temporal fossa. There is another large group in the right occipital region and a solitary lesion just above the left eyebrow.

Exhibition of a Case of Unilateral Papillitis Due to Ethmoiditis

Dr. William Zentmayer presented A. B., female, aged twenty years, single. On April 4 she rapidly lost the vision of the left eye, becoming, she believes, absolutely blind. Twenty-four hours later vision was hand movements, and the field showed an absolute scotoma of about 50 degrees, extending about 30 degrees to the temporal side, 20 degrees to the nasal, 25 degrees above and 30 degrees below. The pupil was partially dilated and reacted sluggishly. The fundus appeared normal.

The possibilities lay between hysteria and retrobulbar neuritis. The Wassermann was negative, and the neurological examination negative except that the history suggested the possibility of hysteria.

Forty-eight hours later there was a papillitis of two diopters, with a great deal of retrobulbar pain, especially on movement of the eyes. The *x*-ray showed a slight clouding of the maxillary sinus. The nasal examination showed pus apparently coming from the sphenoid.

Sphenoidectomy and ethmoidectomy were done. No pus was found. The patient, however, was relieved of all pain by the procedure. There has been a gradual improvement in the vision, so that one week after operation it was 1/30, and the field showed a relative scotoma of about 35 degrees in extent, 15 degrees to the nasal side, 25 degrees to the temporal, 20 degrees above and 15 degrees below. The swelling of the nerve was about one diopter.

The retrobulbar pain and the existence of blindness several days before the manifestation of the fundus changes, and the pain behind the eye and on movements of the eyes, indicate retrobulbar neuritis. The negative physical and laboratory findings suggest spheno-ethmoiditis, and this is borne out by the improvement which occurred after the drainage of these cavities was established, though there was no gross pathological change discovered.

Correction of Cicatricial Ectropion by Overlay

Dr. George H. Cross presented a case showing the reconstruction of the left upper eyelid of a colored man by means of an epidermal overlay. The original injury to the lid and brow was the result of the application of a caustic salve which destroyed the outer surface of the lid, the resultant scar and healing producing a complete eversion of the lid with the margin adherent to the brow.

The lid was separated along the line of attachment and, by blunt dissection, the original external surface was exposed, the lid being put on a stretch by sutures through a piece of adhesive plaster on the cheek. A thin epidermal graft was then taken from the leg above the knee and smoothed into place, then covered with a piece of warm modeling compound and a bandage. The original dressing was removed at the end of a week and a fresh dressing kept on for two days, when all treatment was discontinued. The ectropion was completely corrected and full control of the lid maintained.

Before and After Cataract

Dr. Edwin B. Miller stated that many of us have had difficulty in studying and handling cataract cases before operation, and most of us have more difficulty after the operation has been performed.

In studying a case requiring a cataract operation we have been taught to make a urinary analysis, take the blood-pressure, have smears and cultures of conjunctival secretions made, determine the vision and examine for light perception and projection, to look for focal infections, particularly in the mouth and sinuses.

It was formerly the opinion that patients with a large sugar content had better be left alone, but with the advent of insulin and the proper study and control of diabetes these cases can be safely operated upon. In the presence of a high albuminuric content is is safer to place the patient under a course of treatment and do the operation later.

With the presence of high blood-pressure, 200 or more, there is danger of expulsive hemorrhage and protrusion of the vitreous, not only at the time of operation but also afterward.

Bad teeth and infected sinuses increase the risk, and if one wants to be on the safe side, they had better defer the operation until after these factors are eliminated.

If the pneumococcus, diphtheria bacillus or streptococcus are present in the culture, operation is contraindicated. If a few colonies of staphylococcus or of xerosis bacillus are present, one may go ahead; but if there are many, operation had better be postponed.

The absence of light perception and projection make a cataract operation useless, except where a swollen lens produces the painful symptoms of glaucoma; but even good light perception and projection are not always a safe guide for a favorable prognosis, for lately I have had a case where both light perception and projection were good, but after the operation had been successfully performed we found the patient had pigmentary degeneration of the retina.

The methods of preliminary treatment by drugs are legion, varying from massive doses of bromides to twilight sleep. Most of us have tried many or all of these methods. I believe that in many cases we have done more harm than good, have upset an otherwise good stomach and have caused vomiting and restlessness with protrusion of the vitreous or iris. I am coming to the point where I believe any cataract operation can be successfully performed without preliminary or postoperative drugging.

One should bring the patient into the hospital two or three days before operation, see that the environment is pleasant and the patient comfortable; order the usual cleansing enema; greatly restrict the diet; have the usual laboratory tests made; the toilet of the eye carried out, which consists of scrubbing the face with soap and water and instilling into the conjunctival sac a 25 per cent solution of argyrol every four hours; two hours before the operation, instilling into the eye to be operated on 1 per cent solution of atropine sulphate, repeating this one hour later.

If carefully handled and encouraged, the patient will come to the operating room in good shape, physically and mentally.

I have been using a chart which gives the necessary instructions to the resident physician and the operating-room nurse. I believe it necessary to have at all times a scrubbed-up nurse who has been sufficiently trained to take care of the sterilization of instruments and the preparation of the tray, which in our service is as follows: The table is covered with a quilted sterile pad, over which is spread a sterile sheet; on this we place four sterile trays for the cutting instruments. The first contains C.P. carbolic acid; the second, sterile water; the third, 95 per cent alcohol; the fourth, sterile water. All cutting instruments are placed for five minutes in the carbolic

acid, then passed through the rinsing tray of sterile water, then allowed to remain in the alcohol fifteen minutes and finally placed in the fourth tray of sterile water ready for use. The noncutting instruments are boiled for fifteen minutes.

The instruments to be used are placed on a sterile tray. (There are no unnecessary instruments on this tray; all supplementary instruments are allowed to remain on the sterile table.) The instruments on the tray are placed in a row in the order in which they are to be used, from right to left, so that the assistant can instantly pick up the right one. I believe the surgeon should have a mental picture of just what he will do; of course, there are times when he may have to adopt a different procedure.

The patient is prepared as follows: When coming to the operating room, the eye having been prepared according to the method outlined above, an area near the outer canthus is cleaned and painted with 2 per cent tincture of iodine; a 1 per cent solution of novocaine is now injected at the outer canthus, and while the needle is *in situ* it is slightly pulled back and pointed first to the upper and then to the lower lid. The 4 per cent cocaine solution is now instilled, one drop in the good eye and three drops every three minutes for three doses in the eye to be operated upon. This is followed by three drops of 1 to 1000 adrenalin solution. This allows about ten minutes from the time of the novocaine injection and we have good local anesthesia. Where we have very narrow palpebral fissure, or if the patient is very restless or we suspect fluid vitreous, it may be well to do an external canthotomy. The face around the eye is again scrubbed with green soap and water and the conjunctival sac is flushed with warm boric solution. Four cotton-tipped applicators wet with sterile boric solution are now used: first one, for cleansing the lashes of the upper lid; second, for cleansing the lashes of the lower lid; third, sweeping out the upper conjunctival sac; fourth, sweeping out the lower conjunctival sac. The conjunctival sac is flushed with mercurophen solution, 1 to 8000, then again with warm boric acid solution.

The area around the eye is painted with 2 per cent iodine solution; the lashes are trimmed with a straight scissors, the blades of which are coated with sterile petrolatum, and the lid margins are painted 2 per cent iodine solution and wiped dry with a cotton-tipped applicator; the eye sheet is applied and the patient is ready for operation.

Of the operation for cataract I will say nothing. Every man has

his choice and his own method of doing it. It should present no terror for the man with a steady hand, a little nerve and good instruments.

When the operation is finished the conjunctival sac is flushed with warm boric solution; 1 per cent atropine solution and 25 per cent argyrol solution are instilled. Both eyes are closed and 1000 to 3000 bichloride ointment is spread on the lids; over this is placed a Barraquer dressing, which consists of a thin sheet of cotton, about the size of a half dollar and about ¼ inch thick, wet with warm sterile boric solution; this is pressed down snugly on the eyelids and the hollow space is built up with dry pledgets of cotton until it comes even with the face; over this is placed a sterile pad held down by isinglass plaster; then the aluminum eye-shield is applied to the operated eye and held in place with adhesive plaster; no bandage is used. When the patient is placed in bed, definite orders are given that no drugs of any kind be administered and only liquid diet is allowed for the first forty-eight hours. This is the critical period and a special nurse should be provided; if this is not possible, the nurse on duty should look after the case very carefully.

The following accidents have happened to my cases in the last two years: A patient felt uncomfortable and the resident thought a bowel movement would relieve her, so he gave her two A.B.S. pills and a cup of black coffee. Result, a bad spell of vomiting, expulsive hemorrhage and a lost eye. Another patient wanted coffee and was given it by the nurse, thinking it was all right as it came under the head of liquid diet. Result, vomiting and protrusion of vitreous, with a poor result. A third patient became very nervous and began to vomit. Result, protrusion of the vitreous with protracted healing, a hammock-shaped pupil and a poor result. In this last case I think the sick stomach was induced by the chloral and bromides.

I have discarded these drugs, in fact all drugs, before and after the operation, and now rely entirely during the operation on cocaine instilled into the conjunctival sac and the deep injection of novocaine.

If the operation is carefully done, the toilet of the wound properly carried out and a well-placed Barraquer dressing applied to both eyes, it is not necessary to look at the operated eye for forty-eight hours, when the eye can be opened. If it looks well, I do nothing until the next day, when the eyelids are cleansed with warm boric solution, the conjunctival sac is then flushed with warm boric and

1 per cent atropine solution instilled. If the anterior chamber is reformed, the other eye is allowed open; if not, both are closed for another day. This daily dressing is kept up for a week. The patient is warned about stooping over, as this often opens the wound and causes protrusion of the vitreous or the iris.

Prolapse of the Vitreous. Cut off the protruding part and put on a Barraquer dressing with a gauze bandage, snugly fitting. I tried to improve a case by nipping off the vitreous when it had protruded a second time and did more harm than good. Today I leave it alone after the first cutting and do not mind a gaping wound. I instil 2 per cent mercurochrome and replace the Barraquer dressing and tight bandage and let Nature take care of it. I have been surprised more than once how well she does this. The same is true of *eversion of the flap*, but this can be eliminated by the conjunctival bridge method; I have performed the last four this way, with excellent results. In my experience, the suture method does not prevent expulsive hemorrhage or prolapse of the vitreous or iris, neither does a large conjunctival flap help much after it has occurred.

On two occasions, in my earlier career, I performed cataract operations in the patient's home; in both cases I had trouble, extra work and a lot of worry to get a fair result. Nothing could induce me to do it again. It is a bad practice, I care not how well the home is equipped.

I believe in washing out the débris in the anterior chamber with a properly constructed irrigator filled with warm saline solution, but do not use instruments. One had better allow shreds of capsule and cortex to remain unless it is easily washed out, and later do a capsulotomy, than try to remove it with forceps.

W. H. Bates, in a series of experiments on rabbits, found that when the anterior chamber is first opened, a clear fluid flows out, which does not clot. If, however, a little later the wound is reopened, the fluid which escapes clots spontaneously. Such a clot, when strained and examined by the microscope, shows a structure of fibrin. Each time the eye is reopened the escaping fluid clots like blood after standing for a short time. If the anterior chamber is filled with normal saline solution it prevents the formation of the fibrin-containing liquid and assists in preventing after-cataract.

A cataract operation is so rapidly done that neither the physician, the patient nor the hospital authorities realize the amount of preparation or skill required in its successful performance.

Discussion. Dr. Luther C. Peter said that Dr. Miller's paper opened a broad field for discussion. He wished, however, to call attention to but one point in his paper, namely, the taking of the light projection fields in cataractous patients. Unless great care is observed that the flame of the light of the luminous ophthalmoscope, when used, is kept off the cornea, the patient and doctor may get a wrong impression of the light projection field. Great care should be observed that the light at no time is thrown directly upon the cornea. By observing this precaution a true light projection field will be obtained. It is probable, in the examination made by Dr. Miller of the patient suffering from retinitis pigmentosa, a condition in which we should find very much contracted fields, that the light may have fallen upon the cornea, and thus given the wrong impression as to the patient's light projection.

Iridectomy from the Anatomical Standpoint

Dr. B. Alexander Randall said that the value of iridectomy under various circumstances is too well known to need citation here; but the employment of various substitutes and modifications of the Graefe technic points to frequent clinical shortcomings, while the anatomical study of instances where it has been performed offers valuable explanation of many of its failures. Indeed, the viewpoint of the pathological anatomist is naturally rather pessimistic, since it is almost solely the unrelieved cases that come to his cognizance; unless he shares the view of V. Rokitansky, who said, one day at the Vienna Pathological Institute, "What! no autopsies? Bad medicine."

Yet there are instances where death from accidental or intercurrent causes had brought specimens of supposedly good results under scrutiny; and as it has been my fortune to study some of these, I venture to bring the matters before you.

My competence so to do may be questioned by many of this Section, since I am known to you only in another field than ophthalmology; yet my sectioning of dozens of eyes in the laboratories of Arlt in Vienna, Norris and Risley here, and some from my own practice, has given me an interest and, let us hope, some knowledge of the matter. Yet, rather than to pretend to authoritative dicta, my purpose is to call your attention to the mass of admirable material for study here among us. In the aggregate it probably compares favorably with that which was so beautifully set before you by Fuchs a few years ago; and while we can hardly present any so qualified exponent as the Vienna professor, we ought to have or encourage the making of competent eye-pathologists among us.

The gains and losses by iris excision in cataract extraction may never be justly decided, and the specimens here shown are mainly on the debit side; since it may be claimed to have favored prolapse and incarceration of the iris rather than its prevention. Yet watching, in "simple extraction," the stretching and probable trauma of the intact iris, even where a flap-incision of the corneal margin has been made, inclines one to doubt if the hemorrhage and other defect of sphincter action after iridectomy are not the lesser evils. Even in iridectomies for glaucoma, when a cystoid scar might be expected to give a better "filtration-cicatrix," it is discouraging to find it quite unavailing or even, as in this case, to precipitate, after extraction, a glaucoma that had not seemed previously threatening. Scant circumlental space (Priestly Smith) can hardly be claimed as a factor in increasing the tension after the lens has been removed; and if anterior sclerotomy is often to be curative, we might justly claim at least preventative value from the smoothly healed cataract incision. The most satisfactory clinical achievement of the operation is apt to show in subsequent anatomical study that quite a stump of iris has been left behind; enough fully to block the iris-angle and impede anterior drainage. In this juvenile case we find the iris-root, atrophied to a mere uveal-pigment layer, driven into Schlemm's canal; and although the yielding sclera gave us a buphthalmic axial length of 32 mm., the nerve-head was totally cupped and the posterior pole was staphylomatous, with a wide area of choroidal absorption; glaucoma compelled enucleation.

It is needless now to discuss the various details of these cases, instructive as each of them might be in our study of the many-sided problem; let us hope that it may stimulate the joint investigation of the rich clinical, and especially the anatomical, material in our possession. But it seems pertinent to suggest that in every one of these operations done for the cure of glaucoma we have the unhealed gaping of the impervious Descemet membrane, and therein may be the real value of any such measure. An additional "Fontana space" is thus made for filtration, and its efficacy may depend mainly upon its extent. If so, a large anterior sclerotomy with the Graefe knife, without cutting through the apex of the arc, may prove the most potent measure.

A. Fuchs, in his excellent *Atlas*, illustrates 120 eyes, doubtless the pick of thrice as many. My collection of some 70 eyes duplicates fairly quite one-half of his, some being conditions, Professor

Fuchs told me when here, that he had never seen, such as ossification of the retina. Doubtless Verhoef, of the Massachusetts Charitable Eye and Ear Infirmary, has a still larger collection; but we must among us in Philadelphia have sections of hundreds of eyes, some unique in all likelihood. Our autopsy opportunities of securing postoperative specimens will not often be met, but they may constitute a very precious collection if we shall keep on the lookout for them; for many, doubtless, would obtain them, or have done so, who would be only too glad to place them in expert hands for study and report. Such a committee used to be active, probably still is, in our Pathological Socity, as has, I think, been the case in the Wills Hospital; certainly such efficient agency ought to be doing fine work here in maintaining for our specialty the front-line position that used to be ours, acclaimed by men of judgment just back from a year or two of ophthalmic study in Europe.

Am I only showing ignorance of your current activities, or is there not much that we might be now doing better than is at present the case?

Dr. Richard Bright, an Ophthalmologist's Appreciation

Dr. Burton Chance said that to ophthalmological practitioners any meeting concerning the memory of Richard Bright should be of the greatest interest. While it is probably true that few knew of the varied contributions Bright made to medical science, all constantly have before them the knowledge of his great work on diseases of the kidneys, because of the frequency of the association of disorders of the vision with albuminuria.

Before Bright's day, observers in the beginning of the nineteenth century, and earlier, were aware of and wrote concerning the occurrence of defect or loss of sight in conditions producing dropsy, such as scarlatina, pregnancy and during the puerperium. At about 1837, Bright and his coworkers at Guy's Hospital observed and reported cases of albuminuria in which marked defect of sight had occurred. In a group in whom there were uremic manifestations, loss of sight occurred in four. In certain of the cases the onset of amaurosis was one of the earliest, as well as the most striking, of the clinical features. In the next few years several authors published their observations concerning the occurrence of defects of vision in patients with albuminuria. In 1850, Türck examined microscopic-

ally the retina removed postmortem from a case of albuminuria in which one of the clinical symptoms had been a loss of sight, and he gave the earliest description of the histological changes in albuminuric retinitis. His discovery was soon confirmed by other investigators, and, a few years later, the opinion was put forth by Virchow that the so-called uremic amaurosis was attributable to gross disease of the retina.

Heyman, in 1856, did much to establish the importance of retinal disease in albuminuria; while Zenker's and Virchow's labors dealt chiefly with the pathological anatomy of the retina. To Liebreich belongs the credit of the first detailed description and representation of the ophthalmoscopic appearances of albuminuric retinitis, which he published in 1859, and from which appearances we are enabled to diagnose with certainty the existence of renal disease. These facts do but contribute to the honor of Bright's discovery.

The ophthalmoscope was not invented before 1850. Within the year of Bright's death there were confirmed by the ophthalmoscopic picture of the tissues of the eye the lesions comparable to those which he, hitherto, had seen only postmortem in the kidney, and, by the aid of the ophthalmologist, one was enabled to venture a prognosis as to the duration of life from the visual and retinal changes demonstrable in the living eye.

In this connection we are reminded of Sir William Bowman, who was affectionately spoken of as "the Father of the Kidney." Bowman discontinued his physiological studies to pursue ophthalmology. While Bowman led in that practice (he knew Bright and often must have been in consultation with him), yet he never abandoned his interest in any branch of science he once had studied. We can all imagine the joy which overcame him when he found it possible to view the interior of the eye and there to see vividly lesions associated with the affections of the organ he had for so long examined only after it had been removed from the dead body.

OCTOBER 20

The Mosher–Toti Dacryocystorhinostomy

Dr. Edmund B. Spaeth briefly discussed the function and mechanism of the lacrimal-nasal drainage apparatus. The importance of intact lacrimal canaliculi was stressed.

The indications and the contraindications for and against a dacryocystorhinostomy were considered in greater detail. A short series of cases was presented to compare the end-results with those found following lacrimal cystectomy.

Instruments, technic and postoperative treatment of the Mosher-Toti dacryocystorhinostomy were presented. A very slight modification (rather, individual manner) of carrying out part of this technic was explained.

The paper closed with the statement and the opinion that in properly selected cases a dacryocystorhinostomy is a proper surgical procedure with satisfactory end-results in a high percentage of cases.

Lantern slides were used to illustrate the operative fields and end-results.

Discussion. Dr. Luther C. Peter pointed out that in the treatment of chronic dacryocystitis one must be equipped with more than one type of operation in order to meet the contingencies which arise in clinical work. He said that it is perfectly true, as Dr. Spaeth stated, that following excision of the sac there will be some epiphora. In some instances this symptom is not of very great annoyance, and in others it is of considerable inconvenience to the patient. There are types of dacryocystitis, especially illustrated in the fourth case which Dr. Spaeth presented, in which there was considerable bony obstruction without much inflammatory disturbance, for which the Mosher-Toti operation is better adapted than that of excision.

Dr. Peter said that the technic of the operation is not difficult, and one is not obliged to work with the same meticulous care as is required in excision. In his experience the success of the operation depended upon three points in the technic. In the first place, it is essential that the bony window be made sufficiently large to prevent occlusion after recovery. The second important point in the technic is the preservation of the external wall of the sac and the canaliculi. This is easily accomplished by care in pressing the sac toward the eyeball while the bone is being denuded of its periosteum. A third point, which he was not sure was insisted upon originally by Dr. Mosher, is that of stitching the anterior sac wall and fascia to the periosteum on the nasal side of the wound.

His personal experience with the operation had not been sufficiently extensive as to the number of cases, and not sufficient time had elapsed since any of his operations, for him to speak from personal experience of the ultimate value of the operation. Dr. Spaeth, however, in his paper gave the histories of a number of his patients, one of whom had satisfactory results after the expiration of five years. It is more than likely, therefore, that an operation which has been satisfactory for five years will continue to give satisfactory results.

Dr. Peter regards the Mosher-Toti as the best of the operations which have as their basic principle the draining of the lacrimal secretions into the

nose, and believes it is a distinct contribution to lacrimal nasal surgery, if the cases in which it is used are properly selected. The special indication for its use seems to be the cases in which there is bony occlusion without an accompanying pyogenic process in the sac. When cases are of long standing, and the pyogenic process is therefore marked, excision of the sac is still his operation of choice.

Dr. William Zentmayer said that he had had no experience with this type of operation, as he had been well satisfied on the whole with the results obtained by removal of the sac.

Two Cases of Reticular Degeneration of the Cornea

Dr. William Zentmayer stated that both cases were seen at the Wills Hospital. The case presented was a married woman, aged forty-four years, with six healthy children, none with eye trouble. Parents living; the father probably has corneal dystrophy in one eye. Two members of the family have pterygium.

The patient had recurring attacks of inflammation in the eyes in childhood. On August 2, 1927, the eyes again became irritated, and two weeks later she was admitted to the hospital with superficial corneal ulcers of the left eye.

In the right eye the bulbar conjunctiva is relaxed and inelastic, a narrow fold being pushed up over the lower limbus corneæ by the lower lid. No bulbar injection. The corneal sensibility is diminished. The surface is uneven, but the epithelium is intact.

By oblique illumination there is diffuse haziness throughout, made up of minute grayish lines. By transmitted light they appear as branching lines, forming a network.

Biomioscope examination shows innumerable grayish opacities which, for the most part, are in the superficial layers of the cornea, but some also extend into the middle layers. The opacities are in the form of irregular curved and straight jagged lines of varying diameter, some isolated but mostly branching, with dotlike opacities at their intersections. Where these opacities cross the anterior surface of the corneal parallelopiped, the surface is bulged forward.

Some of the lines can be seen to run from the region of Bowman's membrane obliquely into the middle layers of the stroma. There are two small nebulæ on the temporal side next to the limbus. On the nasal side, at about 3 o'clock, there is a superficial opacity running out from the cornea several millimeters which is covered with superficial vessels from the conjunctival loops. There are no deep vessels in the cornea. The anterior chamber is of normal depth.

The iris is brown, markings distinct. The pupil measures 3 mm. and reacts normally. The lens and fundus seen through the corneal haze seem normal.

The vision in the right eye is 6/12, and in the left 5/60. The tension in both eyes is normal.

In the left eye the cornea shows a similar, but more advanced, condition to that seen in the right eye. There are, in addition, two superficial, centrally-placed ulcers about 2 mm. in diameter, which stain with fluorescein.

The physical examination showed: Kahn test negative. Von Pirquet, negative. Urine: specific gravity, 1028; albumin and sugar, negative; reaction, acid. The nose and throat show hyperplastic ethmoiditis and a chronic infection on the left side. (Operation October 6, 1927.) The tonsils are buried and show a moderate infection. There are several infected teeth.

The second case, which has been too recently seen for careful study, was in a man about sixty-five years of age. The left eye was lost from an injury fifteen years ago. The right eye presented precisely the condition noted in the first case. The surface of the cornea was, however, more uneven, and near the limbus below there was a triangular deposition about 2 by 3 mm., grayish in color, probably hyaline material. There was complete anesthesia of the cornea.

The description as given is from the house notes taken by Dr. Muldoon, of the resident staff of the Wills Hospital.

A Case of Posterior Lenticonus

Dr. William Zentmayer stated that the anomaly was seen in the right eye of a girl, aged sixteen years, with typical pigmentary degeneration of the retina.

By transmitted light, with a +15 D, it had an apparent size of about 4.5 mm., was situated at the posterior pole of the lens and had the characteristic appearance of a drop of oil. When the light was played about it, several very small beadlike elevations were seen near its base. These were only noted with the ophthalmoscope.

The biomoscope study was made by Dr. Cowan, who recently reported on a similar case of Dr. Reese. The 2 cases will be reported in full by Dr. Cowan and Dr. Reese.

Use of Photographic Enlargements for Illustrating Fundus Details

Dr. Alfred Cowan exhibited several fundus drawings to show the usefulness of photographic enlargements for illustrating fundus conditions.

The originals were made with the Nordenson-Zeiss camera and enlarged about four times on semimatte paper. One with very little talent for drawing is in this way enabled to make well-proportioned illustrations of fundus conditions.

The black lines are done with a carbon retouching pencil, and the high lights are scratched with a knife. Colored pencils may be used if desired.

It was found that two enlargements from the same original, when properly mounted, give a very good stereoscopic effect. This is probably due to the slight angular difference made in the retouching.

Gratitude was expressed to Mr. MacMillan, of Street, Linder & Propert, for his help in preparing the photographs.

NOVEMBER 17

Massive Exudate of the Retina

Dr. W. G. Mengel reported a case of retinal disease with massive exudation. He reviewed the literature and found 94 cases reported, including 33 cases classified from the literature by Coats, in 1908.

The case, a boy, aged six and a half years, with right eye involved, had the characteristic yellowish-white mass beneath vessels. The mass at one time appeared similar to circinate retinitis. Vascular changes showing fusiform and aneurysmal dilatations were noted as the condition progressed, and finally the vessels became enlarged and tortuous, approaching the type of the angiomatosis. The case was placed in the Coats' group, 11 cases with vascular changes.

The pathological processes associated were, suspicious tubercular chest lesion with a family history of tuberculosis, and an indefinite right antrum disease.

Discussion. Dr. William Zentmayer said that to the classification of retinal disease with massive exudation given by Dr. Mengel might be added still another, suggested by Nettleship. The description given by him is,

"a class of cases where, in children, gross detachment of the retina occurs in connection with thick plates, sometimes white, sometimes yellow, in or behind it, with an appearance as indicating subretinal hemorrhage."

In a study of cases of massive exudation in the retina one is struck with the difficulty of assigning a given case to any one of the separate classes into which the disease has been divided, as usually one finds, in the same fundus, changes belonging to two or more of the classes.

Some years ago he reported, under the title of "Retinal Disease with Massive Exudation and an Allied Condition," a typical case of Coats' disease and one conforming to the Nettleship type.

A Case of Orbital Growth

Dr. Henry O. Sloane exhibited a patient, J. W., aged nineteen years, who was referred to him on November 14 by Dr. Bloomfield with the following history: About nine years ago his mother noticed that the right eye began to be displaced downward, and this condition progressed until now the right eye is 18 mm below its fellow-eye and is proptosed about 5 mm. He gives the history of having been struck in the left eye when he was ten years of age and attributes his present condition to that injury. The eye was normal until the accident occured. There is no other history of injury or illness.

Examination showed the patient to be a young man, well developed physically. His features were normal in position and appearance, with the exception of the right eye. The right orbit was about 2 mm. below that of the left. The skin of the upper eyelid was reddish in color. The eyeball, together with its coverings (lids, lashes, etc.) was misplaced downward about 18 mm. below the left and proptosed about 5 mm. The rotation of the eyeball was normal in all directions except for some slight limitation upward. The conjunctival vessels were injected; the cornea was clear except for a nebula about 1½ mm. below the center. The pupil was 3½ mm. and reacted normally to light and convergence. The anterior chamber was of normal depth and perfectly clear. The iris was normal. Vision, 20/200. Field studies done at that time were practically normal. There was a slight contraction of the field below and a very slight enlargement of the blind spot.

Under homatropine, the media were clear, the disk margins could be seen fairly definitely, although there was some blurring present. The color was good. There was distinct blurring of the face of the disk due to an edema and a filling of the cup with lymph, which also surrounded and covered the central vessels. There was a fulness

of the veins and a beginning tortuosity of the larger vessels. The region of the macula and the periphery showed no pathological changes.

Palpation beneath the orbit revealed a fairly soft mass, bulging slightly forward. It had a fibrous feeling to the touch. However, it could not be outlined definitely.

Conclusions: A mass, situated beneath the orbit, is pushing the eyeball downward and forward. *X*-ray studies made two years ago at Wills Hospital revealed an infraorbital tumor about 1 inch in length transversely, according to the patient's statement. As to the nature of this growth, one can only speculate at this time. It may be either a mucocele, angioma or, as Dr. LeFever has suggested, a cold abscess. Operative procedure has been advised and will be carried out as soon as complete studies are made.

Discussion. Dr. William Zentmayer said that last spring he operated upon a case which resembled the one shown by Dr. Sloane, a boy of about the same age with a history of a protrusion of the eye of ten years' standing, said to have followed a blow.

The condition was almost exactly the same except that the downward dislocation was not so great. He was treated with *x*-rays, radium and large doses of potassium iodide for a period of one year without appreciable improvement.

The pathologic report was a sarcoma, with a large amount of fibrous tissue.

Dr. Zentmayer said he had sometimes noted that irrespective of the position of the growth there was often a palpable mass beneath the superior orbital margin. He thought that this might be due to dilatation of the orbital veins and changes in the orbital tissues.

Dr. E. A. Shumway said that all would agree that orbital growths, such as in Dr. Sloane's case, were among the most difficult conditions to diagnose that came before the ophthalmologist. As Dr. Zentmayer had said, the surface character of the growth to palpation did not constitute a criterion as to its real consistency, as the real tumor might be further back in the orbit and be covered by edematous tissue. The presence of whiplike cords on the anterior surface in Dr. Sloane's case suggested neurofibroma, but in such cases there was usually involvement of the eyelids. The negative findings to the *x*-rays would exclude osteoma, and the negative Wassermann, if corroborated, would similarly exclude gumma. The very slow development of the growth was evidence of its benignity, and would rule out round-cell sarcomas or epithelioma growths invading the orbit from the neighboring sinuses; and the absence of increased leukocytosis would preclude an inflammatory process. In the majority of cases positive diagnosis can be made only by microscopical examination of the exenterated tissue.

and it might prove to be a slowly developing sarcoma arising from the periosteum or other connective-tissue elements in the orbit, or a growth arising from the optic nerve sheaths.

Comparative Study of the Rôle of Pigment in the Physiology of Vision

Dr. Francis Adler spoke of the rôle of pigment in the physiology of vision, pointing out that each of the various types of pigment present in the mammalian eye shows analogies in structure and function with the various pigments found elsewhere in Nature. The functions of light protection, light isolation, photodynamic sensitization and phototactic movements so frequently met with in pigments in Nature are also seen in the fully developed eye. The pigment cell is a necessary component of every photoreceptor.

A Lantern Demonstration of Tumors of the Conjunctiva

Dr. E. A. Shumway and Dr. Perce DeLong gave a lantern demonstration of microscopic slides illustrating tumors of the conjunctiva from Dr. Shumway's collection.

The growths included tyloma (hypertrophy of the epithelium); papilloma; papillary carcinoma and epithelioma of the limbus, with involvement of the eyeball and peribulbar tissue; cysts of the lid margin; keloid of the cornea; subconjunctival osteoma and lipoma; dermoids of the limbus; nævi of the conjunctiva; benign cystic epithelioma; lymphosarcoma and sarcoma (melanotic and alveolar).

Dr. Shumway commented on the clinical appearance of the growths, especially the differences between carcinomata and sarcomata, and the marked delay in involvement of the eyeball by the latter, and spoke briefly of his theories of the etiology of dermoid growths and nævi. He showed also papilloma, carcinoma and cystic adenoma of the lacrymal caruncle, and a case of ophthalmia nodosum (due to caterpillar hairs) and one of tarsal growth in vernal conjunctivitis. Many of the cases had been reported before the American Ophthalmological Society and the College Section on previous occasions.

An Unusual Form of Symmetrical Lenticular Opacities in the Adult Nuclear Zones

Dr. Luther C. Peter observed this condition in a young man, sixteen years of age, during the course of an examination for glasses.

In the equator of the adult nuclear zone there was an irregular line such as we find in a pulse tracing, the tracing forming a complete circle as viewed with the ophthalmoscope. Under high magnification, with the 3A objective on the slitlamp, the tracings were found to consist of an irregular line such as one finds in taking an original pulse tracing, T. There were no other evidences of lenticular opacities and the condition was present to the same extent in both eyes.

Inquiry as to the history in the case revealed the fact that the patient suffered from malnutrition during the first few years of his life. There was no history of tuberculosis in the family and the patient at this time is well nourished. The slitlamp examination definitely showed that the irregular line was immediately outside of the nuclear zone. It is probable, therefore, that these opacities developed in the first few years of the patient's life and should probably be regarded as the result of malnutrition. The lenses in other respects are entirely normal, as shown by the slitlamp.

The case is placed on record because, in his experience, no similar case has been found in his slitlamp studies, and although a complete review of the slitlamp literature was not made, he was unable to find any reference to a similar case. The condition undoubtedly is nonprogressive.

An Instrument for the Removal of Intraocular Foreign Bodies

Dr. T. B. Holloway exhibited an instrument designed for the removal of intraocular nonmagnetic foreign bodies. The principle of the instrument was based on a type of bronchoscope utilized in the clinic of Dr. Chevalier Jackson at the University Hospital. For use with the fluoroscope (the ingenious method described by Dr. Cross at the last meeting of the American Ophthalmological Society) it would require a change in the character of the handle.

Discussion. Dr. George H. Cross said that it was rather difficult to discuss many points of Dr. Holloway's new instrument for the removal of nonmagnetic foreign bodies from the vitreous, as it was as yet untried. He had no doubt that Dr. Holloway had discovered how difficult it was to manipulate any instrument under a fluoroscopic plate, especially so if it is complicated and at all lengthy.

In his very limited experience with the removal of nonmagnetic foreign

bodies from the vitreous, the simpler and less complicated the instrument and method the more likelihood there was of success. One is always pleased to have the benefit of a new type of instrument when operating upon these extremely difficult cases, and we will all be pleased to hear from Dr. Halloway after he has had the opportunity of using his new instrument.

In closing, Dr. Holloway stated that he wished to reaffirm that in his statement to the effect that this new type instrument would probably produce less disturbance of the vitreous than the one used by Dr. Cross, he was in no sense reflecting upon the last-named instrument. The suggestions for this instrument had been given to the instrument maker with the intention of using it in the case of a child with a small particle of brass or copper in the eye, but, unfortunately, the foreign particle could not be satisfactorily seen with the fluoroscope.

Moving Pictures of Patients with Certain Orbital Conditions

Dr. T. B. Holloway exhibited a series of moving pictures showing various types of orbital disease. These were exhibited before the Section, not with the intention of demonstrating the cases selected, but simply to show the availability of well-selected cases for the moving-picture demonstration. Still further, with the ease with which good results can be obtained, it was felt that these would be invaluable for teaching purposes. In most institutions, when approaching such studies as orbital diseases or the external ocular muscles, it is frequently difficult to assemble a sufficient number of cases to illustrate the various types of conditions. Equipped with a reel of moving pictures, one always has at hand what might be regarded as a "canned clinic."

DECEMBER 15

Symblepharon from Molten Metal—Plastic Repair

Dr. Burton Chance exhibited a case of plastic repair in a case of symblepharon following molten metal burns, consisting of dissection of the cicatricial mass by Arlt's method; ligation of restricting bands, and covering the raw surfaces with a graft from the superior cul-de-sac.

Ptosis and Paralysis of the Superior Rectus

Dr. Chance exhibited a case of complete ptosis and paralysis of the superior rectus of the right eye, believed to have been caused

during birth. It was relieved by a tucking advancement of the superior rectus muscle, tenotomy of the inferior rectus, and Hess' operation upon the lid.

Fibrous Material on the Optic Disk

Dr. Chance also showed a case with obscuration of the optic disk by a mass of fibrous material, probably formed after extravasation.

Hemorrhage of the Sheath of the Optic Nerve Occurring in the Fellow-eye after a Ten-year Interval

Dr. George H. Cross reported this case on account of the interesting and unusual circumstances in which a hemorrhage in the sheath of the optic nerve of the right eye, with total loss of vision, was followed after a ten-year interval by a hemorrhage in the sheath of the nerve of the left eye, causing total blindness.

H. L. M., aged fifty-one years, merchant, was a patient on March 23, 1911, for refraction, at which time vision in each eye was 20/15. On December 19, 1914, at 8 P.M., the patient states that he started to lose his vision in the right eye about ten o'clock in the morning and by 4.30 P.M. everything was black; he had some slight pain in the eyeball. Two days previously he walked into the door-jamb, striking the right eyebrow; yesterday he helped lift a barrel of chickens into a wagon and lifted a barrel of turnips out of a wagon. He was not in pain and only came to the office following the insistence of members of his family that he obtain medical advice.

Examination. Right eye: Pupil, 5 mm., round, reacts slightly and very sluggishly to light. Ophthalmoscopic examination: Media, clear; disk, round; margins, sharply defined; color, good; a large physiological cup present; bloodvessels somewhat full but not swollen or tortuous; macula, normal. There were no hemorrhages or areas of pigment disturbance noted. His blood-pressure was 183 mm. Vision, no light perception.

December 22, 1914. Disk margin somewhat blurred; no other changes noted.

December 26, 1914. Serous edema of the retina, manifested by a swelling surrounding the disk.

December 29, 1914. Consultation with Dr. Radcliffe and Dr. Schwenk, who both concurred in the diagnosis of hemorrhage in the sheath of the optic nerve in the right eye.

About ten years later (to be exact, May 5, 1924), while on his farm, he ran up a hill, yelling, to chase some cows out of a cornfield; suddenly objects started to blur and he started toward his house, reached the porch calling for help as he could not see to get about. His family doctor stated his systolic pressure at this time was 260 mm. On examination, his left eye revealed a similar condition to the right eye except that he claimed to be able to distinguish a very bright light.

Patient stated that he had severe headache for many years, and several weeks before this attack, while driving a horse along the road, he suffered a temporary loss of vision which later cleared up so that he could see to drive home; also he had numerous nasal hemorrhages. Eight weeks before his death, which occurred on April 26, 1927, he had a tremendous nasal hemorrhage.

Acute Unilateral Optic Neuritis, Toxic in Origin

Dr. G. Oram Ring presented a report of a case of acute unilateral optic neuritis (retrobulbar) of toxic origin.

The patient, male, aged thirty-six years, had been in perfect health, but had noted a slight blurring of vision about one week previous to Dr. Ring's examination, the blur just preceding his visit being so dense as to suggest impending blindness. The right eye was normal. In the left there was a questionable light perception with swelling of the nerve head two diopters above the eye-ground, with no other fundus lesion. Light response was sluggish in the affected eye. The only urinary findings consisted of the presence of oxaluria and a too high specific gravity. The blood count revealed a leukocytosis (11,250) with an approximately normal differential. The Wassermann reaction was negative. An *x*-ray of the teeth condemned a left central incisor, which was extracted, and upon culture was found to be infected with hemolytic and viridans streptococci. Both tonsils showed the presence of the same organisms, the hemolytic predominating. The tonsils were promptly removed. Sinus *x*-ray showed some thickening of the ethmoid cells of the opposite side; but clinical examination revealed a marked septal deflection with middle turbinal pressure on the affected side, but no clinical signs of sinus disease. As a precautionary measure the septum was resected so that the sinuses could be opened on the left side if the response to the other measures was

not pronounced. The examination other than that noted above was negative. An autogenous vaccine was promptly given and continued until 20 cc. had been administered. The elimination was guarded from the skin, kidneys and intestine. As the toxemia was removed the vision began to return, and upon the date of its report had improved to 20/30, with almost normal field.

Reference was made in the paper to the fact that in the author's experience the toxic manifestations in association with disease of the teeth were most usually in the uveal tract, less frequently noted as a sclerokeratitis, occasionally as a fugacious episcleral congestion and rarely as an optic-nerve involvement.

Discussion. Dr. Judson Daland opened the discussion, and was followed by Dr. William Zentmayer, who said that without intending in any way to question the etiologic diagnosis made by Dr. Ring in his case, he thought that in drawing a conclusion from the results following the removal of the focus of infection, we should bear in mind that the prognosis in acute retrobulbar neuritis is on the whole favorable, many of the cases tending to recover under general treatment.

It is because of this well-recognized course of the disease that there has been, on the part of European Continental clinicians, a slow acceptance of the views held by American ophthalmologists as to the part played by focal infections and the results attributed to their removal.

He said that it should not be construed from this that he did not favor the immediate removal of foci of infection which the examination might disclose, but he would not necessarily draw the conclusion that this was the cause of the improvement. Recently a young woman had come to him at the Wills Hospital complaining of rapid loss of vision in the right eye. The vision equalled counting fingers at about 1 meter. Between the time of this visit and arrangements being made for the removal of her tonsils, which were the only discoverable source of infection, the vision had already improved to 6/60 under the administration of sodium salicylate.

In nearly all cases of retrobular neuritis, even where the visual function of the eye has been recovered in full, a pallor of the papilla is observed, which is permanent.

Dr. Luther C. Peter stated that it seemed important that the other phase of this question should be presented. He was entirely in accord with the promptness with which the essayist acted in determining the etiology of the infection in the case which he reported. He was convinced that the residual atrophy and visual disturbance which one finds in these cases is in direct relation to the promptness with which operative procedures are instituted in cases of this type. This is particularly true when the infection arises in the nasal sinuses.

Several cases which he had had under his care recently illustrated this point. The first case was a woman who lost her sight overnight, but

delayed a whole week before she consulted her physician, and at least twelve days elapsed before operative interference could be practiced. The sphenoids were badly diseased, but, notwithstanding operation, total loss of vision was sustained. A second case was that of a medical student whose central vision became blurred during the course of three or four days. He made a prompt recovery without any residual atrophy after opening up of the posterior ethmoids. A third case was that of a young woman whose vision was lowered in one eye, and in whom operative interference was not practised for several weeks after the onset of the visual disturbance. In this case there was a definite residual atrophy with fairly full fields and fairly good recovery of central vision. The final case is one which had been under his care during the past three weeks, in which the ocular lesion is one of papilledema of about two dipoters. This patient developed a marked contraction of the peripheral field and a cecocentral scotoma extending well beyond the point of fixation. Following operation on the sphenoids the patient made a rapid recovery, the peripheral fields are back to normal and the cecocentral scotoma is now reduced to but a small island beyond the point of fixation.

It is true that many of these patients show a residual atrophy with what seems to be fairly normal fields, but a careful study, especially by colored test-objects, demonstrates the fact that the fields do not fully recover and central vision rarely becomes entirely normal. This relation, therefore, between the residual field disturbance and the time of operation becomes an important one, and it emphasizes the necessity for prompt action in determining the source of infection and, if possible, eliminating the same.

Dr. Ring, in closing, expressed the definite conviction that the toxemia resulted in his case from the teeth and tonsils, and said that the septal work was precautionary. He advised taking nothing for granted, and advocated hunting for, and eliminating, every discoverable source of toxemia.

Diseases of the Eye and Adnexa Due to Fungi and Higher Bacteria

Dr. Sanford R. Gifford, of Omaha, Neb., pointed out that conditions suitable for the growth of fungi of higher bacteria are met with in the conjunctival sac or on the cornea, and a number of these organisms so prevalent in Nature have been reported rarely as causing various ocular conditions.

An organism resembling the leptothrix, which forms delicate unbranched threads, has been found in sections of Parinaud's conjunctivitis. The author reports finding a similar organism in 2 out of 3 cases. He also found leptothrix in cultures in a case of chronic conjunctivitis and in several cases of chronic infection of the Meibomian glands.

Streptothrix or actinomyces, forming delicate threads showing

true branching, is the cause of concretions of the canaliculi. This condition is a fairly common one, the reporter having seen 7 cases, in 2 of which pure cultures of actinomyces were obtained. All cases cleared up rapidly when the canaliculus was slit and the concretion removed. In these cases actinomyces acts as a saphrophyte growing in the canaliculus, but other cases are reported in the literature in which it has acted as a true pathogenic agent, forming ulcers, membranous conjunctivitis and orbital cellulitis.

A brief review is given of the literature of infections around the eye by sporotrichosis, which are especially common in the Mississippi Basin. In Omaha, Dr. Gifford has seen 6 cases, in 1 of which a pure culture was obtained, and the reporter has seen 3 cases with pure cultures obtained in 2. Diagnosis by cultures is valuable in these cases, since the use of potassium iodide internally and locally nearly always brings about a cure.

The reported cases of infection of the cornea with aspergillus, the usual cause of keratomycosis, were discussed. The author reports 1 case which resulted in panophthalmitis and showed sections of the cornea in this case. The importance of bacteriological examinations which will reveal occasional infections by these unusual organisms was emphasized.

Discussion. Dr. Fred D. Weidman said the work of the Giffords deserves some special form of recognition. Blastomycosis is Chicago disease; coccidioidal granuloma is California disease, and, in conformity with medical traditions, it is quite proper to henceforth designate ocular sporotrichosis as Omaha disease.

One thing that dermatology and ophthalmology have in common is the visibility of their fields. Dr. Weidman could envisage how that in ophthalmology as in dermatology the specialist took particular pride in an appreciation of the connections between the eye and internal medical states, and that he was always on the alert to detect some such connections. He could also understand how the purely local conditions could also present problems in diagnosis which would draft the utmost diagnostic resources of the ophthalmologist and which would, all things considered, be even more fascinating than the internal medical phases of eye diseases. Fungus diseases of the eye embraced both of these phases, some being part of a systemic mycosis, whereas others were local. What he had to say would be confined to the latter.

As to personal experience, he had studied but two lots of material from the eye. Both had been received from Dr. Shumway. One was a fresh culture from a lacrimal calculus, and the other was an old, dried-out specimen of lacrimal calculus. In the former, after much study, he had finally

come to the conclusion that the thread organism concerned was not a streptothrix or leptothrix but a "bacillus in chains." In the second case nothing grew in the culture, but on direct examination made from teasing of the specimen the classical filaments of actinomyces were found, and the case may have been very well one caused by Actinomyces foersteri. It was necessary to study streptothrix materials *in vivo* (hanging-drop preparation) before finally concluding as between the bacillus in chains and some of the thread organisms. Ordinary bacteriologic smears would not do. He showed slides from the calculus illustrating the true branchings of Actinomyces foersteri and the Gram-positive granules which studded the filaments.

Turning next to Dr. Gifford's presentation, the classification of the fungi was notoriously mixed up, and in a short discussion like this it was useless to attempt to include that phase of the subject.

It would be necessary to exercise caution in respect to leptothrix infection as seen in sections, because elastic fibers sometimes simulated these non-branching filaments. Concerning concretions in the tarsus, there was also reason to exercise caution before finally concluding that they were of fungus causation, because there were such extensive fatty foci in this situation. Frozen sections, stained by Sudan III, would disclose that the tarsus normally is extensively and diffusely infiltrated by fat, and these circumstances favor the infiltration of lime salts. As to aspergillus infections, it was not common to find the fruit-heads in tissue; aëration seems necessary to bring them out. In autopsies on parrots at the Zoological Garden the forms found in the caseous mesenteric lymph nodes were exclusively mycelial, whereas in the lungs the classic fruit-heads could be seen extending into the air sacs.

Of course, iodine was the great standby in fungus infections anywhere in the body. Dr. Weidman could understand how, in the case of the eye, the urgency might be so great as to require the most radical iodine therapy. In some cutaneous mycoses the French were employing Lugol's solution intramuscularly (10 cc. to the dose), or even intravenously, mixed with serum. He doubled very much that such extreme measures were commonly indicated in dermatology. Dr. Gifford had rendered a real service to the audience; he was a pioneer in his subject and had brought much not alone to ophthalmology but to special mycology.

Dr. William Zentmayer wished to recall to Dr. Weidman's memory a third case of a calculus of the canaliculus which he studied and on which he was kind enough to give him a report and some beautiful photomicrographs. The case was that of a colored women, aged fifty-six years, who came to the hospital because of a swelling over the upper canaliculus, accompanied by a mucoid discharge from the punctum. The condition was of two years' duration.

A calculus was removed. It was a spheroidal, yellowish-white, stony body about 4 mm. in size. The surface was finely granular and homogenous. A study of the mass was made by Dr. Weidman, who reported that the calculus was composed practically entirely of the threads of a streptothrix, such as has been frequently described in this class of cases. Cultural studies were negative, probably because the calculus was kept dry for a

long time. The photomicrographs presented show the character of the growth.

Dr. Edward A. Shumway said that one of the specimens of lacrimal concretion mentioned by Dr. Weidman was from a patient of Dr. Zentmayer's. Dr. Shumway had reported 2 cases, but in only 1 was a pathological examination made. In this case a bacillus in chains was reported by Dr. Weidman which has not been definitely identified, and Dr. Shumway thought the clinical condition was due to the presence of this organism. In ordinary cases the concretion produces no destruction of tissue locally, but in this patient there was a cavity back of the canaliculus which connected with a fistulous opening in the conjunctiva in the cul-de-sac of the lower eyelid. In the second case the concretion was in the upper canaliculus and produced no destruction of tissue. Its removal was followed by prompt cure of the dacryocystitis.

Retinal Arteriosclerosis with Intermittent Vessel Changes

Dr. Luther C. Peter and Dr. Edmund B. Spaeth pointed out that there are several points of interest in this case. The case itself is not unusual, but it illustrates the changing character of the retinal disturbance which may be observed, and the photographs are offered as a means of recording and preserving permanent records for future comparison.

The case is one of the nephritic type, with high blood-pressure, the systolic being well over 200 mm. Hg. and as high as 312 mm. The diastolic pressure is well over 100 mm. usually. A branch of the superior temporal artery shows complete occlusion, and the inferior temporal branch presents an unusual white sheath for some distance from the disk. Ten days ago the entire inferior artery was similar to the superior and there was a definite contraction of the central nasal field. There are evidences of arteriosclerotic retinitis in both eyes, to follow Foster Moore's classification, although one would naturally classify the case as one of retinal arteriosclerosis of nephritic origin. The exudates and the hemorrhages are characteristic of arteriosclerotic retinitis, but the changes in the vessels suggest the nephritic origin of the retinal arterial disease.

The second case, which was presented only by photograph and visual field, is equally instructive, and illustrates the second group of Foster Moore's classification, namely, arteriosclerosis of the retinal vessels. The superior temporal was suddenly occluded about fourteen months ago. The vessel is quite small and the blood stream for two disk diameters beyond the disk is quite irregular. It is surrounded by thickened white media. The field shows the extent of retinal dysfunction.

All the arteries are exceedingly small. The veins are large and dark, and are definitely indented and even lost when crossed by arteries. There are no hemorrhages present, but the few exudates which are visible are characteristic of the type to which this case belongs. The blood-pressure in this case is 160 mm. Hg. over 80 mm.

A Case of Massive Phlegmon following Novocain Injection in a Diabetic

Dr. Luther C. Peter and Dr. Edmund B. Spaeth presented a case of bilateral intracapsular lens extraction for senile cataract. The extractions were done after preliminary iridectomies.

The extraction in the left eye was entirely uneventful. Before the left lens extraction, blood-sugar was as high as 160 mg. per 100 cc., though it was normal the day before and the day of the operation. Extraction was done under cocaine instillations, and with novocaine injections under the conjunctiva at the limbus at the site of the usual conjunctival flap and at the outer canthus to block the filaments of the facial nerve supplying the orbicularis palpebræ. Recovery was normal.

The lens extraction operation for the right eye was done about six weeks later. The day before the operation, blood-sugar content was 105 mg. per 100 cc. For some reason the blood-sugar content was not taken the day of the operation. The same technic was used at this time as was used for the left eye. The extraction was most uneventful. Three hours after the operation the patient complained of great pain over the external canthal angle. This had to be quieted with morphine. The next morning there was a tremendous swelling of all the tissues over the external canthal angle, with a very slight temperature rise. Blood-sugar that day was 360 mg. per 100 cc. The first dressing was done twenty-four hours later, a total of forty-eight hours after the operation. Swelling had continued for this interval until the right face was at least one-third larger than the left side of the face. The swelling was brawny red, hot and with very tense skin. It was impossible to open the lids. Without further anesthesia the upper and lower lids were incised throughout their entire length down to the fascia, and the outer canthus from the angle externally for 2 inches. The subdermal tissues were a pale gray color, showing a sharply demarcated line of delineation from the more nearly normal superimposed dermal and epidermal

tissues. There was no suppuration, however, though some blood-tinged serum exuded from the cut phlegmonous tissues. When the lids were opened it was seen that the area of injected conjunctiva was also involved in this gangrenous process, though here some suppuration had started. The pupil was dilated, anterior chamber reformed, and the cornea and iris seemed normal. Twenty-four hours later, suppuration had started in the gangrenous tissue. For several days this tissue could be seen sequestrating itself from the normal tissues above and below it. After seven days the suppuration had advanced so extensively that radical incisions were made in the face, cheek and temporal scalp, Dakin's tubes inserted and the case treated with Dakin's solution irrigations.

The affected conjunctiva sloughed off *en toto* without damage to the eye or without nullifying the results of the lens extraction save for one small corneal nebula and for some irregular astigmatism of the cornea, both of which are disappearing rather rapidly. After four weeks the patient was discharged with the recovery seen here. Two weeks later he was readmitted with a gangrenous ulcer over the os calcis, following a small blister from a new pair of house slippers. This also is recovering rapidly.

The blood-sugar, which had reached 360 mg. per 100 cc. twenty-four hours after the operation, dropped to as low as 76 mg. per 100 cc. At that time the patient had two mild attacks of insulin shock. The blood-sugar would swing from 200 mg. per 100 cc. to less than 100 mg. per 100 cc. within twenty-four hours under insulin therapy and dietary control. It was most difficult to establish a dietary-insulin-blood-sugar balance in the case. During the recovery period the patient's condition was an infallible index of the state of his blood-sugar. As this went up in its vagrancies, so also did the patient's temperature, pain at the site of the phlegmon and the amount of suppuration ascend.

The problem now remaining is a simple one of plastic surgery. Much of the subcutaneous tissue has been lost, as can be seen, through the subsequent slough with much scar-tissue deposition, but it is by no means of great difficulty. No epidermal tissues were lost in the late suppurative process, it was purely subdermal. This one factor will assist greatly in the later correction.

The entire case illustrates rather too well the danger of tissue damage in diabetic conditions. With but little doubt, sterile water would have caused the same degree of tissue reaction and damage

as was here caused by the novocaine injection, though the chemical reaction novocaine has to protoplasm did perhaps increase the damage in this case. The outstanding fault in the case was the failure to determine blood-sugar content on the day of the operation, even though that of the previous twenty-four hours was within the normal limits.

Discussion. Dr. Edward A. Shumway said that while what he had to say was not exactly germane to the subject of Dr. Spaeth's communication, he thought it worth while to bring to the attention of the members the importance of careful blood-sugar estimation before operating on a diabetic patient. In a recent case of cataract extraction in which he had relied upon the report of the family physician, operation was followed by persistent vomiting which lasted for two days. The blood-sugar was tested and found to be over 400 mg. per c.mm. Fortunately, the ultimate result was good, but consultation with Dr. Petty brought from him the dictum that the upper limit of safety for any operation was 155 mg. in the blood in a diabetic or potential diabetic. Dr. Shumway thought that it was important for all operators to bear this in mind.

Traumatic Iridodialysis with Macular Changes

Dr. Frederick Krauss presented a patient with rupture of the iris, associated with macular and other changes. He had been shot in the right eye with a piece of tinfoil. The concussion produced a large hemorrhage in the anterior chamber. Upon its absorption, there was found a rupture near the outer rim of the iris, a large Vossius ring on the lens and a decided wrinkling of Decemet's membrane. There was a marked neuroretinitis. A few weeks later the macula was seen as a large hemorrhagic spot surrounded by spokelike radiations in the retina. These were visible with red-free light. The vision remained low, the field greatly contracted, with a large absolute scotoma to the temporal side of fixation.

PROCEEDINGS

OF THE

SECTION ON OTOLOGY AND LARYNGOLOGY

JANUARY 19

Salivary Calculus of the Submaxillary Gland Associated with Infection

BY DR. ARTHUR WAGERS
(By Invitation)

About 60 per cent of salivary calculi are found in the submaxillary gland. The calculi composed essentially of calcium phosphates and carbonates are considered to be secondary to infection. Years may pass before these calculi manifest any symptoms. The case of an adult female who had a stone in the right submaxillary gland associated with severe local infection was presented. The calculus was visualized by *x*-ray and removed surgically under local anesthesia. Reference was made to the proximity of the lingual and hypoglossal nerves to the submaxillary gland and the necessity for using care in order to avoid injury to these structures.

Discussion. Dr. George B. Woods referred to several cases of submaxillary calculi in which he had surgically removed the stones.

Dr. Harry A. Rothrock mentioned the importance of the symptoms of sublingual pain and fullness at the time of eating due to the pressure of retained saliva.

Dr. George M. Coates cited a case of submaxillary calculus that was so mobile that its removal was quite difficult.

Dr. Karl M. Houser spoke of an adult colored male who experienced recurrent blockage of Wharton's duct. On three occasions the duct was dilated and each time a branching yellow cast of the duct was expressed. This cast microscopically was amorphous, showing neither cells nor crystals. Relief of sublingual discomfort immediately followed the removal of the cast.

Dr. Arthur Wagers, in closing, spoke of another case in which the stone had been spontaneously expressed from Wharton's duct.

Peripheral Paralysis Resulting from Focal Infection

BY DR. WALTER L. CARISS
(By Invitation)

Two cases of peripheral paralysis resulting from focal infection were reported. The first was that of a colored baby with a right-sided mastoiditis and a left facial palsy. The left ear-drum was slightly inflamed, but otherwise the ear and mastoid of that side were negative. Both mastoids were operated upon and both found to be greatly necrosed and filled with pus. Complete recovery of the paralysis followed within six weeks.

The second case was that of an adult male who, nine days before admission to the Presbyterian Hospital, began to experience numbness of his lower extremities. This condition was rapidly progressive, so that in a few days he was unable to move his legs or hips and had lost most of his arm power. A diagnosis of multiple neuritis was made. Complete study and examination were negative except for clouding of the left frontal, ethmoid and maxillary sinuses.

An open operation of these sinuses under ether anesthesia was followed by the patient's discharge from the hospital on the twenty-first day apparently completely recovered. Three years have elapsed since this procedure and to date the patient has had no recurrence.

Discussion. Dr. Nathan P. Stauffer called attention to the necessity of thorough study of all cases of peripheral neuritis in the attempt to discover infective foci.

Dr. George M. Coates spoke of a case of facial paralysis in an adult male that recovered following mastoid operation. Little evidence of mastoid disease was present.

Opening the Bite According to the Wadsworth Technic of Articulation and its Effects upon the Temporomandibular Area

BY F. G. HAWSWORTH, D.D.S.
(By Invitation)

Dr. Hawsworth believes that many cases of ear disturbance, such as deafness and tinnitus, are secondary to a retrogression of

the mandible. This commonly occurs in middle life and following the loss of teeth. A muscular imbalance is produced, resulting in a backward thrust of the mandible, encroaching upon the auditory canals. This idea was first brought forth by Dr. George S. Monson, of St. Paul.

Habitual retrusion of the mandible causes pressure and gradual resorption of the interarticular cartilage and the surrounding joint structures, which may include part of the tympanic plate. Backward pressure may result in almost complete closure of the aural canal, together with continued pressure and irritation of the tympanic plate. The above observation is that of Dr. Walter Henry Wright, of Pittsburgh.

A line drawn from the center of the pupil to the line of the lips (mouth closed), should equal a line from the nasal septum to the mental process; if the latter line is shorter than the former, imbalance is present.

Such cases are treated by the fitting of artificial dentures, which gradually restore the mandible to its proper position. Sometimes this treatment requires eighteen months for completion and the production of four to six dentures.

Discussion. Dr. Thomas Aiken asked the procedure to be followed when imbalance is present with a full complement of teeth.

Dr. George B. Wood asked if any changes had been observed in the aural canal.

Dr. George M. Coates asked which aural cases should be considered proper cases for such appliances, and how a differential diagnosis was established.

Dr. Benjamin Shuster felt that the glenoid fossa was so far beneath the ear structure that it was very doubtful if the disturbance mentioned would influence hearing or tinnitus.

Dr. Hawsworth, in closing, said that if a full complement of teeth is present, but imbalance present, correction is brought about by the application of crowns or cusps. He himself has not seen encroachment or closure of the aural canal.

A Review of the Year's Work on Laryngeal Tuberculosis, by Dr. George B. Wood

(See *Archives of Otolaryngology*, December, 1926, p. 527)

Discussion. Dr. Curtis Eves finds the cautery most useful in selected cases of laryngeal tuberculosis. The quartz-light tubes hold some promise of being of value.

Dr. Gabriel Tucker has recently done a tracheotomy in 1 case of laryngeal tuberculosis for dyspnea. Remarkable improvement followed. Radium or x-ray will probably produce chondritis or perichondritis.

Dr. Philip Stout has used tracheotomy in about 12 cases. In no case was there regret that the operation had been performed. In his cases tracheotomy has frequently relieved dysphagia. Sun-baths and injection of the superior laryngeal nerve with alcohol are both of value.

Dr. George B. Wood has seen no relief of dysphagia after tracheotomy and uses this operation only for the relief of dysphea.

FEBRUARY 16

Otogenic Pathology in Paralysis of the Abducens Nerve

BY DR. W. H. SEARS
HUNTINGDON, PA.

(By Invitation)

The conclusions set forth by Dr. Sears were based on the study and analysis of 210 cases of sixth-nerve paralysis due to aural suppuration: 190 cases found in the literature since 1910, 16 unpublished cases from the records of other otologists and 4 cases observed by the author.

Almost twenty years ago, and again three years later, Gradenigo brought forth his now-famous syndrome characterized by the symptom triad—suppurative otitis, severe temperoparietal pain and homolateral abducens-nerve paralysis, subsequent to the diffusion of purulent infection from the tympanic cavity to the apex of the pyramid.

In 81 per cent of cases the suppuration is acute. In most of the cases reported the tympanic membrane perforated spontaneously, or if myringotomy was performed it was done late in the disease. Rarely did the tympanic membrane remain intact.

With aural abducens palsy, pain is almost always present. In 162 cases it was absent 3 times, in 10 it was slight to moderate, and 148 times it was severe. The pain usually was homolateral and was relieved very little by narcotics or anodynes. Frequently it persisted for days. Very often there is associated ear pain, which disappears after adequate tympanic drainage is established. The most frequent pain site was the frontotemporoparietal region.

The abducens palsy is frequently sudden and complete, sometimes

gradual. The first subjective symptom is homonymous diplopia observed by the patient on looking toward the affected side. This may, in the presence of intense pain, pass unnoticed. Objective findings are a marked convergent squint and inability to rotate the affected eyeball outward beyond the midline.

It is important to bear in mind the intimate anatomic relation of the Gasserian ganglion and the sixth nerve, for a pathologic process influencing one can quite conceivably involve the other. Direct extension of the tympanic infection may be through peritubal pneumatic cells or perilabyrinthine cells. A pneumatic pyramid is essential. This extension has taken place indirectly through the tegmen, through the floor to the jugular bulb, anteriorly and mesially to the carotid canal and plexus, through the labyrinth and posterior to the antrum and mastoid cells.

In the series of cases studied there were 42 deaths; 31 were due to meningitis. Many slides were shown demonstrating the anatomic features of the temporal bone which influence the occurrence of this symptom-complex.

Discussion. Dr. George M. Coates states that about twenty-four years ago Gradenigo brought out his symptom-complex, the pathology of which was demonstrated by Perkins in 1910. Since then it has been recognized that various ocular palsies may be otogenic in origin. Egleton has found 4 cases of sixth-nerve palsy occurring in cavernous sinus thrombosis. The gravity of this symptom should be impressed on the otologist. Dr. Sears' series shows a 16 per cent mortality. Many recover following such simple procedures as myringotomy or mastoid surgery. Conservative surgery at other times is not enough. The variations of Dorello's canal and the possibility of the nerve being pinched in this canal are most interesting. Recently Dr. Coates has seen a case recovery following mastoid operation.

Dr. J. Clarence Keeler stated that this syndrome most often occurs in late spontaneous perforation or myringotomy. This calls for early interference. Most often the streptococcus is present. Recognition of this condition is becoming more general. Some cases develop the conditions some time after the acute ear condition has subsided, 4 were as late as four months after operation.

Dr. Temple Fay (by invitation). The difficulty in explaining how an infection of the pyramid selects the sixth nerve adjacent to it, though lying free in the subarachnoid space, and the extension occurring to the fifth nerve at times, appears difficult. However, if we analyze the pain manifested in these cases, we find it is not true trigeminal pain and is not truly confined to the trigeminal field. The pain has much of sympathetic origin, both in the trigeminal and in the adjoining fields, and the painful points seem to follow the arterial distribution.

The explanation, in the light of what Dr. Sears has pointed out regarding the structure and anatomy of this region, would appear to implicate the carotid artery in its passage through and below the involved region; inflammation traveling along the sheath of the carotid, reaching the sixth nerve, which lies adjacent to it in the cavernous sinus, and involving the sympathetic fibers given to the Gasserian sinusion, as well as the posterior mesial aspect of the ganglion itself.

Slides and cases were given to illustrate the type of sympathetic pain encountered. Dr. Sears' contribution goes a long way toward explaining certain indefinite pains of sympathetic origin and the possible involvement of the carotid artery in its canal, and as a constructive piece of work Dr. Sears' paper is a monograph in character.

Dr. Alexander Randall has seen a number of bilateral and unilateral cases of sixth-nerve palsy. When bilateral they are usually specific. The extreme vulnerability and strength of the sixth nerve must be remembered. In a large series of mastoid cases pachymeningitis was found in about two-thirds of the cases. Since this is so one would expect sixth-nerve involvement when the petrous is invaded by suppuration.

Dr. Luther Peter asked Dr. Sears what per cent of cases recover without operation. In most of the cases seen by Dr. Peter a mastoid operation had been performed sometime before. The ophthalmologist does not consider the paresis alone an indication for surgery, but stresses early operation where the optic nerve shows any sign of involvement. Gradenigo's syndrome may be masked by other conditions.

Dr. Mathew Ersner has had 1 case where sixth-nerve palsy was relieved following removal of an impacted tooth. In typical cases he has always found a large cellular mastoid. In 1 case where the mastoid cavity was filled with bismuth paste the symptom-complex appeared.

Dr. Robert J. Hunter has seen Gradenigo's syndrome in basilar meningitis, the meningitis being verified at autopsy. In another case it was present in otitic brain abscess and disappeared following abscess drainage. Some have mistaken a previously existing strabismus for a recent sixth-nerve palsy. Bárány maintains that peripheral palsy of the sixth nerve can be differentiated from central paralysis by caloric and turning tests. Pain occurs in many cases of facial palsy, probably due to involvement of the geniculate ganglion and nerve of Wrisberg.

Dr. Sears felt that conjugate deviation might arise from meningeal irritation. In the series reported, 20 cases were not operated on; of these 4 died.

MARCH 16

Otitic Meningitis

BY DR. E. B. GLEASON

Prompt myringotomy and early mastoid operation, if necessary, are the best measures of avoiding otitic meningitis. Many cases

of acute otitis media showing cervical rigidity and other symptoms of meningitis promptly recover when these things are speedily performed. In cases that do not so respond but develop a true meningitic process, the disease begins as an osteomyelitis, with infection of the meninges as soon as, or before the aural mucosa. Most all fatal cases of aural intracranial complications terminate as septic leptomeningitis.

Two clinical varieties of leptomeningitis can be differentiated; the serous or protective and the purulent or infective. The finding of bacteria in the spinal fluid establishes the diagnosis of the latter variety. The treatment of the serous type is rest in bed, ice-cap and the diminution of intracranial pressure by purgation with magnesium sulphate. Repeated lumbar puncture is dangerous, as it may spread infection. Urotropin in 20-grain doses t.i.d. is advisable.

Otogenic leptomeningitis usually begins as an extradural abscess. At its outset it is a localized infection of the cerebrospinal fluid and meninges. Surgical treatment consists in removing the source of infection, aided by appropriate medication and diet. The identity of the organism in the spinal fluid should be learned and serum therapy inaugurated in certain types. Optochin intraspinally has been used by some in pneumococcus infections. Ventricular or cisternal puncture in connection with lumbar spinal puncture has been advocated and used by some for the purpose of washing out the subarachnoid space with saline or other solutions.

Discussion. Dr. Francis Clark Grant stated that Kolmer has recently reviewed the subject of the use and value of intraspinal solutions, sera, etc., in meningitis. He concluded from this study that neither chemicals nor antibody solutions, so used, favorably influence the disease if used alone. If they can be used along with adequate surgical drainage, life can be prolonged and saved in some cases. This was experimentally demonstrated on dogs. Dandy advocates cisternal drainage in staphylococcic and streptococcic meningitis. In the pneumococcic types the pus is too thick for efficient drainage. As almost all cases are fatal Dr. Grant believes that radical measures are justifiable and advocates immediate surgical drainage. Chemo- and serum-therapy should be used in conjunction with the surgery.

Dr. Robert J. Hunter has applied the methods of Kolmer and Eggleton to several cases. Saline irrigation from ventricles to cisterna was performed in 1 case, which gave temporary improvement. In 2 other cases the advice of Dr. Grant was followed. Wide mastoid operations were performed, the labyrinth removed and the cisterna drained in this region. All cases ended fatally. The danger of early surgery lies in the difficulty of establishing

an absolute diagnosis of meningitis. Only when bacteria are found in the spinal fluid is this certain. Cases of meningismus recover following mastoid operation alone. Very probably many cases of meningismus would become true meningitis if operation were not promptly performed.

Dr. S. MacCuen Smith considers that the prevention of otitic meningitis is best accomplished by early myringotomy and mastoid operation, to get rid of the infective focus. Lumbar puncture readily establishes the diagnosis of meningitis. In his experience, cisternal drainage, repeated lumbar puncture, intraspinal urotropine, etc., have all been of no value when infective meningitis is once established.

Dr. George M. Coates stated that thorough eradication of the focus of infection is most important. All diseased mastoid cells, as well as those that may become diseased, should be removed. Some cases that show symptoms of meningitis recover following mastoid operation. These are probably localized. If radical surgery is performed before bacteria appears in the spinal fluid, then this operation may have been done on a case that would have recovered after more conservative measures. Eggleton advises carotid ligation in order to put at rest the affected parts.

Dr. Gleason feels that we are approaching better results in the treatment of otitic meningitis.

Bronchial Cyst Encroaching upon the Larynx Causing Fatal Suffocation

BY DR. W. R. WATSON

Dr. Watson exhibited a pathologic laryngeal specimen in which there was a bronchial cyst which apparently was an integral part of the larynx, a portion of which formed a "ball-valve" glottic obstruction. The cyst entered the larynx in the right ventricle and extended downward to lie over the vocal cords.

The patient had been admitted to the Episcopal Hospital as one of bronchial asthma. Her first respiratory trouble began six months before admission, at which time she was told she had asthma. She was at this time relieved by hypodermic medication. These attacks became more frequent and severe, until she was finally sent to the hospital as an atypical case of bronchial asthma. Speech was normal. On admission she was cyanotic, but not laboring for breath. It was noted, on chest examination, that inspiration would proceed normally to a certain point, then suddenly stop. No signs of asthma were found. The patient was admitted at midnight and died the following morning. No laryngeal examination had been made.

Discussion. Dr. Fielding O. Lewis stated that often patients with laryngeal pathology are treated over long periods of time as cases of bron-

chial asthma. Laryngeal stenosis and asthma should always be differentiated. In the case cited, if seen early, a tracheotomy could have been done and later followed by appropriate laryngeal surgery.

Unusual Manifestation of Mastoid Disease with Lateral Sinus Thrombosis

BY DR. AUSTIN T. SMITH
(By Invitation)

In December the patient, a male, aged twenty-six years, suffered from a head cold and pain in the left ear. At the end of a few days he had apparently recovered, except for a hissing noise in the affected ear which persisted for several weeks. About five weeks after his initial symptoms he developed severe pain in the left ear, associated with some mastoid tenderness but no aural discharge. The left auricle was pushed forward, a diffuse external otitis was present; temperature, 99.2° F., and *x*-ray evidence of bone destruction and probably perisinus abscess of the left mastoid. This condition improved so much that operation was postponed for ten days. At that time, however, pain and swelling returned.

At operation there was found subperiosteal pus, which had escaped from the mastoid *via* the emissary vein. Much bone destruction, thrombosis of the lateral sinus and a large perisinus abscess was found. The antrum was sealed off by sclerosis and was opened with difficulty. The pus on culture was found to be sterile. Appropriate surgery was performed and uneventful recovery followed.

Discussion. Dr. Robert J. Hunter referred to the case of a child that developed acute otitis after a fall. An occipital mass was present that proved to be subperiosteal pus that had escaped through the emissary vein. This vein was followed until free bleeding was obtained, where it was ligated. The sinus was not disturbed. Recovery was uneventful.

Spontaneous Rupture of the Lateral Sinus

BY DR. HORACE J. WILLIAMS

Dr. Williams presented the case of a child, aged five years, on whom a mastoid operation had been performed. The sinus had been

exposed at the time of operation, on account of softening of the sinus plate. The sinus itself was not thrombosed. The wound was drained by a small rubber tube, which was placed in the antrum and brought out the inferior angle of the opening. The skin was closed by metal clips. On the fourth day the skin was healed and the clips removed. On the seventh day a profuse hemorrhage occurred, blood escaping around the tube and lower wound angle. The skin incision was immediately opened and bleeding controlled by packing. At the end of eight days the packing was permanently removed with no further bleeding. Recovery was uneventful.

Discussion. Dr. Robert J. Hunter: Spontaneous ear hemorrhages, as reported in the literature, are rarely attributed to rupture of the sigmoid sinus. Very often, where aural hemorrhage does occur, the internal corotid artery is involved. Politzer states that all corotid ruptures are fatal, but probably some of the ear hemorrhages that recover are really of corotid origin. The rupture of the corotid usually occurs at the bend. In such cases it would be possible to ligate the corotid in the neck and again above this bend by opening the mastoid cavity.

Dr. Fielding O. Lewis had seen a patient with tinnitus and slight deafness. This tinnitus was relieved by corotid pressure. With an auscultation tube in the ear a bruit could be heard. This bruit could also be heard with a stethoscope in the temporal side. On *x*-ray examination the outline of a possible intracranial growth was discovered.

Dr. George M. Coates once saw a case similar to the one reported. This case, however, had unrecognized jugular-bulb thrombosis and the hemorrhage was possibly due to pressure from above. Then, too, a drain in contact with the sinus wall may have caused traumatic erosion.

APRIL 20

Some Neurological Complications of the Ear, Nose and Throat

BY DR. T. H. WEISENBURG

(By Invitation)

Dr. Weisenburg has for eight years given lectures to students of otolaryngology in the Graduate School of the University of Pennsylvania. During that period he has found a vast number of the conditions of the ear, nose and throat which show points of neurological interest.

The association of optic atrophy, optic neuritis and choked disk with nasal conditions is important. Some rhinologists consider this sequence to be quite common. Others deny this frequency.

Sluder considers the optic nerve to be involved by direct extension, by hyperplasia of the soft parts or by pressure following osteitis or periostitis. Not all cases can, however, be so explained, for often no sinus pathology can be found yet sinus surgery improves vision. Perhaps these cases are due to toxic influences.

Retrobulbar optic atrophy is more common than optic neuritis. This can follow sinus infection without direct involvement of the optic nerve, probably by toxic influences. Primary optic atrophy never follows sinus disease.

Dr. Weisenburg does not consider that sinus disease can or does produce a frank choked disk. Some observers have reported choking to the extent of eight diopters. Dr. Weisenburg does not feel that more than two to three diopters ever occurs, this being due to optic neuritis.

The procedure of operating on the sinuses for optic neuritis before a thorough neurological study has been made is to be condemned. Many brain-tumor cases are brought to the brain surgeon in which nasal surgery has previously been performed.

Pain in the face may be due to involvement of the fifth, ninth, nasopalatine ganglion or the sympathetic system. Dr. Weisenburg's experience with sphenopalatine ganglion pains has been disappointing in that, while cocainization has temporarily alleviated the pain, alcoholic injection of the ganglion has failed to do so. The nasopalatine pain as described by Sluder does occur, but the syndrome is uncertain. This may be due to variations in the ganglion, to which Sluder has called attention. Cushing and Frazier doubt the existence of this syndrome.

Inflammatory processes of the middle ear, sinuses or nose may give rise to thrombophlebitis of the dural sinuses, meningitis, extradural abscess and abscess of the brain. About 90 per cent of brain abscesses follow chronic infections.

The diagnosis of brain abscess is most difficult. The history must be carefully considered, the clinical course followed and frequent neurological studies made for the detection of signs of localization.

Vestibular tests should always be made, as an aid in the diagnosis of an intracranial lesion. They are not infallible and the diagnosis should not be made upon these tests alone.

Vertigo may be toxic, epileptic or functional in origin. In the experience of the speaker, most vertigoes have proved to be toxic and have a good prognosis. It is difficult, but important, to distinguish between aural vertigo and the minor types of epilepsy.

Encephalitis affecting the vestibular nuclei in the medulla may give rise to labyrinthine vertigo.

Discussion. Dr. Wells P. Eagleton (by invitation): The future of meningitis may be considered bright. At the beginning all cases are curable. If at this time adequate surgical drainage can be provided, meningitis of a purulent nature can be prevented. All efforts to cure general leptomeningitis have failed.

The chronological history is of great value in the diagnosis of brain abscess. Chronic temporosphenoidal lobe abscess is to be differentiated from cerebellar abscess by (1) visual fields and (2) ventricular puncture. In other than pontile-angle lesions the vestibular tests are of little localizing value. These tests are of value in detecting increased intracranial pressure of the posterior fossa, trauma (especially hemorrhage) in the posterior fossa, toxic conditions and disseminated sclerosis. In all cases of vertigo, turning and caloric tests should be made. If the nystagmus duration is reduced, they should be considered cases of focal infection. Arteriosclerosis and sexual causes may give rise to vertigo. Dr. Ross Hall Skillern considers the toxic possibilities most important in retrobulbar neuritis. Focal infection, direct extension of infection, hyperplastic sinus conditions, toxic influences and negative pressure must all be considered. Surgery, providing adequate aëration and drainage, must be performed on apparently normal sinuses in many cases of retrobulbar neuritis. Improvement is due to ventilation and the relief from toxemia. If there is a rapidly progressing optic neuritis, operate at once. Dr. Skillern's experience with sphenopalatine ganglion symptoms has been disappointing.

Dr. Louis Clerf was impressed by the fact that the larynx featured but little in the report of the neurologist. Many times a sensation of foreign body in the throat is due to the lingual tonsil. Laryngeal pain is most often referred to the ear. Phonic spasm, congenital stridor of infants, infantile laryngeal paralysis, laryngeal vertigo, nervous cough and functional aphonia all have a neurological background. In paralysis of the recurrent laryngeal nerve the cause of the paralysis is sought elsewhere.

Dr. Nathan P. Stauffer asked when surgery should be used in purulent meningitis.

Dr. George M. Coates asked Dr. Eagleton his procedure in sterile meningitis without localizing signs.

Dr. Eagleton stated that surgery should be used when there is localized infection.

Dr. Weisenburg said that every otologist and rhinologist should have some knowledge of ophthalmology. He should be capable of reading eye-grounds and taking visual fields. Many times the vestibular tests show evidence of cerebellar tumor when toxic conditions, such as alcohol, lead or syphilis, may be the causative agent. In all functional diseases the investigation of sexual conditions is important.

OCTOBER 19

Difficulties in the Diagnosis of Lateral Sinus Thrombosis

BY DR. NATHAN P. STAUFFER

Dr. Stauffer reported 6 cases of mastoid infection that developed symptoms of sinus thrombosis subsequent to operation: 4 of these cases were proved to be the result of abscess and thrombosis of the lateral sinus by operation on that vessel; in 1 of these cases the use of a spinal-fluid manometer during the compression of the jugular veins gave positive proof of the existence of thrombosis; in another the test was negative when a sinus thrombis was present; in 4 cases the test was not used; 1 case developed metastatic abscesses; 2 cases recovered without operation on the sinus; 1 of these cases had developed thrombophlebitis of the veins of the leg which at first symptomatically simulated sinus thrombosis, the other was probably due to inflammation of the petrous portion of the temporal bone.

Dr. Stauffer concluded from his experience that the manometer test may be of great aid in the diagnosis of doubtful thrombosis cases. In several cases repeated blood transfusions were used with splendid results.

Discussion. Dr. Karl M. Houser mentioned a case of sinus thrombosis in which the manometer test (Ayer-Toby) was diagnostic and of great value.

Dr. Douglas McFarland considers blood transfusion to be of decided value in these cases.

Dr. W. L. Cariss considers the Ayer-Toby test to be very useful in the diagnosis of sinus thrombosis.

Rhinorrhea following Rasping of the Frontal Sinus

BY DR. E. W. COLLINS

(By Invitation)

After two applications of a rasp to the left nasofrontal duct the patient, in the case reported, developed profuse rhinorrhea.

Although no chemical tests were made to establish with absolute certainty that the fluid from the nose was cerebrospinal fluid, there seemed to be little doubt that such was the case. Complete recovery followed after eighteen days of rest in bed and palliative measures. X-ray examination of the skull showed wide vessel margins, suggestive of increased intracranial pressure. It was the opinion of the author that at the time the rasp entered the frontal sinus there was a herniation of the brain through an opening in the posterior wall of the frontal sinus.

Atypical Esophageal Stricture in an Infant

BY DR. JAMES A. BABBITT

A newborn babe began to vomit as soon as it began to nurse. This vomiting could not be relieved by atropine, gruel feedings or any of the usual palliative remedies. Partial atresia of the esophagus was diagnosed and a gastrostomy considered. The intern on service, however, succeeded in introducing a small tube into the stomach by the nasal route. The baby did so well on nasal feedings that gastrostomy was not necessary. The diagnosis was confirmed by roentgenograms.

Studies in Vienna and Zurich

BY DR. ROBERT J. HUNTER

Dr. Hunter spoke informally of his recent experiences in European clinics.

Atypical Neuralgia

BY DR. TEMPLE FAY

(By Invitation)

This term applies to those types of pain about the face and head which cannot be classified as true trigeminal neuralgia. Many consider pain of this sort to be psychic, because no adequate explanation is available. Dr. Fay feels certain that pain of this sort does occur and considers the following possible pain pathways:

Seventh, ninth, tenth, eleventh, twelfth cervical nerves and sympathetic. Although there is evidence that the facial nerve does carry sensations of deep pressure, it is very doubtful if it can be responsible for the transmission of pain after the section of the sensory root of the fifth nerve. Two cases were operated on by Dr. Fay and the results in these cases demonstrated that the ninth does not carry pain fibers, and that its sensory function is devoted to the sense of taste and vasomotor responses secondary to the gustatory reflex. There is considerable evidence to show that the eleventh and twelfth nerves are purely motor. Dr. Fay then concludes that, if facial pain exists after section of the sensory root of the trigeminal nerve, it must reach the cerebrum by the vagus, posterior cervical roots or sympathetic.

In order to eliminate still further, the speaker, in a case of facial pain resulting from carcinoma, sectioned the sensory root of the fifth, the intracranial root of the ninth, and the upper two posterior cervical roots. The patient still suffered from deep aching pain in the ear, and pain in the face and neck of the operated side. Anesthesia was complete over that side of the head and neck except a small area within the concha of the ear, which is supplied by the vagus. Autopsy of this patient, fifteen months later, left no doubt as to the complete destruction of the nerves mentioned. As a result of this case the conclusion was advanced that pain must travel by either the sympathetic or vagus. In one case of carcinoma the vagus was sectioned intracranially. This section relieved all pain in the ear, throat and tenderness to deep pressure along the upper aspect of the neck. In another case, section of the hypoglossal relieved pain behind the ear, the lateral aspect of the neck and tonsil region. This was probably due to the vagus branch which the hypoglossal receives. In the case in which the vagus was cut the sympathetic chain had been interrupted below the superior cervical ganglion, leaving the vagus only as the pathway by which pain impulses could possibly reach the brain. It is possible that fibers associated with the vascular tree may be carried along the carotid and gain entrance to the cord *via* the cervical or thoracic segments. In order to prove or disprove this idea, observations, including sympathectomy, must be made. From the observations of Dr. Fay he feels that these pain sensations of atypical neuralgia are probably carried by sympathetic fibers to the vagus and by that nerve trunk reach the brain-stem.

Discussion. Dr. William A. Sears (by invitation): Dr. Fay has called attention to a form of intractable neuralgia arising in suppurative otitis and associated at times with paralysis of the sixth nerve. This pain is atypical and is far removed from the suppuration in the temporal bone. Dr. Fay's suggestion of the transfer of a tympanic infection to the sheath of the carotid artery as it passes through its canal in the petrous portion of the temporal bone is quite possible. Its further extension along the sheath to the Gasserian ganglion and sixth nerve seems equally possible. This mechanism probably does serve to explain the pain sometimes present in jugular thrombosis.

Some years ago Weisenberg reported a case of glossopharyngeal pain. Dandy has reported 2 cases in which the ninth nerve has been sectioned. I suggest that Dr. Fay, in closing, discuss the so-called glossopharyngeal neuralgia.

Hunt has studied the facial nerve with special reference to a sensory field, using cases of otitic herpes zoster. He assumes this condition to be the result of involvement of the geniculate ganglion, which is similar to a posterior spinal-root ganglion. The neuralgia associated with these cases is often severe. I have had 6 of these cases under my personal care: 4 of these cases had complete facial palsy and 2 had sixth-nerve palsy associated with facial paralysis. Otalgias and mastoidalgias may arise from carious teeth, ulceration of the tongue, pyriform fossa or pharynx.

Although time is lacking to discuss in detail the Sluder syndrome, cocainization of the sphenopalative ganglion has given unquestioned relief in a large number of variously located pains.

Dr. Francis C. Grant said that he has had a somewhat different experience from that of Dr. Fay. Many cases of facial neuralgia have not been relieved by section of the sensory root of the fifth nerve. Injections of the sphenopalative ganglion have not been very successful. Carotid stripping and section of the sympathetic did not give relief. Out of 16 cases so treated, only 1 was relieved. The work of Dr. Fay is throwing more light on the subject, and the selective nerve cutting in cases of carcinoma is entirely justifiable.

NOVEMBER 16

Dr. Robert Lukens demonstrated an instrument called the demonstroscope. This is a device which makes possible the simultaneous viewing of the larynx by two observers in addition to the operator.

Certain Laryngeal Conditions

BY DR. WILLIAM V. MULLEN

(By Invitation)

The tendency of the present is to accurate physical and pathological laryngeal diagnoses, but to neglect the physiological aspects

of the larynx. Adduction and abduction are confusing; constriction and dilation would be better terms. More notice should be given the accessory respiratory muscles in the performance of these functions. The works of Negus and Mosher have proved that the epiglottis does function in the act of swallowing, and Dr. Mullen has observed that dysfunction follows its removal.

The recurrent laryngeal nerve is practically never involved by thyroid disease unless that disease is of a malignant nature. This is accounted for by the fact that the laryngeal nerves are well protected in the groove in which they pass to the larynx.

Slides were shown illustrating the variations of laryngeal enervation. Dilworth found that the inferior branch of the superior laryngeal nerve may pass through a foramen in the thyroid cartilage and may have a close anastomosis with the recurrent laryngeal nerve. The arytenoidus muscle is supplied by both the superior and laryngeal nerves. Negus emphasizes that this muscle is concerned in closing the larynx. Probably this is a big factor in glottic closure after section of the recurrent laryngeal nerve. It is probably not quite correct to consider the superior laryngeal nerve entirely sensory with the exception of the motor fibers to the cricothyroid muscle. Any injury or stimulation of the recurrent laryngeal nerves brings the cords to the midline, where they remain until atrophy of the muscles takes place. The term "cadaveric position" is a misnomer, for the cords do not take this position after death. Dilworth considers the laryngeal nerves as a highly specialized vagus plexus especially assigned to the larynx.

Different observers have obtained different results from experimental stimulation of the recurrent laryngeal nerves. Some have obtained cord adduction, others abduction. These variations were due to different strengths of stimuli. Dr. Mullen feels that conclusive work of this type must be performed on monkeys, as the dog larynx varies too much from the human to allow comparison. Some workers have obtained splendid results by anastomosis of the recurrent laryngeal with the phrenic. Reverse results have here, too, been secured. This seems to be due to a lack of control of the amount of stimulus applied.

Three cases were cited. In the first, no relief of closure of the glottis was obtained by ligation of the recurrent laryngeal nerve in a woman who had tight closure of the cords after thyroidectomy. In the second case the vagus was ligated during a thyroid ligation.

In this case the cord went to the outerside. A third became aphonic the day after thyroid operation. Both cords were found to be midway between abduction and adduction. Esophageal paralysis, too, was present. Recovery followed after feeding ten days by means of a tube. In addition to the above cases, 3 cases were mentioned that experienced pulmonary atelectasis after recurrent laryngeal nerve injury during thyroid operation.

Discussion. Dr. Albert T. Brubaker spoke in reference to the respiratory function and its manner of performance. There must be little resistance offered to air in its passage into the lungs. Phonation depends on cutting off the upper portion of the larynx from the lower. The vocal membranes must be widely separated to allow the air free passage. The area of the open glottic chink is about 160 sq. mm., which is less than the area above and below. Therefore, there is some resistance offered at this point and this resistance varies as the square of the above area. The posticus muscle is much larger than the laterals. This gives rise to the erroneous idea that more power is required to turn the vocal processes out. It does nothing more than separate the vocal cords, and the large size is not necessary. But the vocal membranes, in order to prevent suffocation, must be abducted at all times. This probably explains the large size of the posticus muscle. Only certain parts of this muscle work at certain times, during which periods the unused fibers recover from fatigue.

Each muscle must have its own enervation from special nerve cells. In the upper portion of the medulla is that group of cells which acts as the respiratory center. In addition, the larynx constantly receives impulses through the recurrent laryngeal nerve for the purpose of maintaining adequate breathing space. The lateral cricoarytenoid muscles, as well as other phonatory muscles, have their own nerve centers. When the recurrent laryngeal nerve is split the posterior crico-arytenoid muscles can be stimulated without affecting the laterals. Confusion has arisen only because the nerves to the various muscles run side by side in close physical union. The arytenoid muscle receives its nerve supply from the superior and recurrent laryngeal nerves of both sides. In the etherized dog the effects of stimulation vary, depending upon the depths of anesthesia and the strength of stimuli.

Dr. Charles H. Frazier spoke concerning the neurosurgical aspects of the larynx. The tracheal collapse of literature does not occur, but is due to glottic closure following nerve injury. If the inferior thyroid artery is clamped close to the thyroid capsule the nerve is not in danger. It is most likely to be injured at the isthmus, giving rise to immediate or delayed laryngeal paralysis.

In 1924, Jackson had unsatisfactory results in dilation and ventricular cordectomy for the relief of laryngeal paralysis. At this time, Dr. Frazier performed a nerve anastomosis in a number of cases. The descendens noni was joined to the stump of the recurrent laryngeal. In all cases the tone

of the cords returned, but in none was the improvement sufficiently good to allow decanulation. In the last 4 cases operated upon the phrenic nerve was used. Three of these cases were anatomically successful; in the fourth the recurrent laryngeal nerve could not be found. So far, 18 cases in all have been so treated. In 12, the descendens noni was used, in 4 the phrenic. Sixteen cases were anatomically successful; 8 were improved; 6 not improved, and 2 very much improved.

Dr. Fielding O. Lewis stated that in 2 cases of carcinoma, where he had removed the epiglottis, swallowing was accomplished without difficulty. There was trouble at first in training the patients to swallow, but after a few days, with a feeding tube, they were able to take food fairly well. In 1 case where radium was used the nerves of deglutition were paralyzed. The course of the laryngeal nerves is long, and hence they are exposed to numerous dangers. Often the laryngologist may be of signal aid to the neurologist.

Dr. George B. Wood referred to the removal of the epiglottis in tuberculosis of the larynx, *in toto* or in part, either by the cautery or the disease itself. In these cases he has not seen the swallowing function damaged. If the fibers passing through the aryepiglottic folds are not disturbed, dysfunction will probably not occur.

Dr. Robert J. Hunter called attention to the idea that the human epiglottis is is more or less a vestigial structure. In the ruminant it allows simultaneous eating, breathing and smelling.

Obstructive Laryngeal Dyspnea

BY DR. GABRIEL TUCKER

The dyspneic patients in whom the cause of the symptoms is laryngeal obstruction show characteristic signs on examination that at once distinguish them from other forms of dyspnea in which there is no obstruction to the larynx. The characteristic sign is indrawing at the suprasternal notch, around the clavicles, the intercostal spaces and the tip of the sternum on inspiration. These signs are particularly apparent in children with obstructive laryngeal dyspnea. Cases of this type require surgical treatment and very often require emergency tracheotomy or intubation. The treatment of the other types of dyspnea is essentially medical and is usually not so great an emergency. The administration of morphine and other sedatives in obstructive laryngeal dyspnea, because of its sedative effect on the accessory respiratory muscles and the inhibitory effect on the cough reflex, increases the retention of secretion and tendency to pulmonary edema. The use of sedatives is, there-

fore, inadvisable in obstructive laryngeal dyspnea. The early recognition of the signs of indrawing and the patient's inability to sleep are the indications for surgical relief of the dyspnea. The safest and best treatment for the relief of obstructive laryngeal dyspnea is tracheotomy below the first tracheal ring, under local anesthesia.

Tracheotomy was done in a case of obstructive laryngeal dyspnea due to postdiphtheritic cicatricial laryngeal stenosis in which the signs of indrawing were marked. The dyspnea was entirely relieved, with the disappearance of the indrawing. This report was illustrated by a film of very instructive motion pictures.

DECEMBER 21

Clonic Spasm of the Muscles of the Eustachian Tubes

BY DR. KARL M. HOUSER

(By Invitation)

Politzer, Schwartz, Brunner, Todd and Boeck have observed and described the above condition. Such cases are rather infrequent, and a search of the literature of the past twelve years produced no cases of this sort.

The patient to be presented is a male, aged twenty-four years, who for the past eight months has experienced annoying clicking sensations in both ears, more marked upon the right side. The onset of this condition was gradual and not accompanied by an acute cold or sore throat. Neither tonsillectomy, tubal catheterization nor bouginage has given any relief.

This clicking sound on the right side can be heard by an observer at a distance of 4 feet from the patient. On the left it is barely audible with the auscultation tube. No movement of the drumhead can be seen when the clicking sound occurs, nor is there any relation to the pulse-beat. If a bougie is inserted into the Eustachian tube for a distance of 1.5 cm. the clicking sound on that side is no longer present. The sound reappears as soon as the bougie is withdrawn. With the nasopharyngoscope it can readily be seen that a contraction of the muscles connected with the anterior lip of the Eustachian tube accompanies each clicking sound.

Discussion. Dr. George M. Coates mentioned a similar case, where this clicking could be heard 10 feet from the patient. In this case, movement of the muscles of the soft palate and movement of the drum occurred with each click. The bougie here had no effect upon this sound. In another case of ear suppuration an audible click could be stopped by movements of the jaw.

Dr. B. Alexander Randall spoke of an auditory click that was present in a female who had chorea. The origin of the click is probably from the separation of the moist surfaces of the tube.

Dr. E. B. Gleason said that he had his only case of this sort about twenty years ago. This case improved under bromides.

Dr. S. MacCuen Smith said that cases of objective tinnitus aurium are infrequent and their etiology has seldom been determined. We are indebted, therefore, to Dr. Houser for the report of his interesting case, and, more especially, his definite information as to the etiology, as the clicking ceased when he introduced the Eustachian bougie.

About twenty years ago I was chairman of the Section on Otology and Laryngology of the American Medical Association, and Professor Gustav Killian, of Frieburg, Germany, was the guest of the Section at that time. Following our meeting at Atlantic City, the Section was invited to Philadelphia to hear Professor Killian lecture on foreign bodies of the respiratory tract and their removal. On the following day the members were invited to participate in some clinics at the Germantown Hospital, at which time I presented a case of bilateral objective clicking tinnitus aurium which could be heard about 3 feet from the patient. Professor Killian's assistant, Dr. Koellreutter, suggested that it might be stopped by the introduction of a catheter and bougie. This he tried on both sides, without any relief, so a different set of muscles was evidently responsible in this case.

Among others, this case elicited considerable interest on the part of Dr. Alfred Stengel, who went to considerable trouble and expense in the perfection of apparatus to determine the cause of the patient's objective tinnitus. The patient became so impressed with her importance, from the fact that we had shown her on various occasions before medical meetings, that she finally demanded from Dr. Stengel and myself a substantial pecuniary reward "if we expected to learn anything more about her trouble," as she said, and she therefore passed from our observation without our being able to further investigate the cause of her tinnitus.

Demonstration of Tapia's Artificial Larynx

BY DR. FIELDING O. LEWIS

In two patients who had had laryngectomies performed, this artificial larynx was demonstrated. This apparatus, which is quite simple in construction, permits the development of reasonably clear speech after some weeks of practice.

A Study of the Effect of Ceanothus Americanus on the Coagulation Time of the Blood

BY DR. A. A. S. GIORDANO AND DR. DWIGHT L. WILBUR (By Invitation)

The above drug was tested on a group of 62 medical students. It was found that for this group the average normal coagulation time, when estimated by capillary tubes, was five minutes, fifty-four seconds. The observers concluded from their experiment that:

1. The alkaloids of Ceanothus americanus definitely reduce the coagulation time in the normal individual. In the series tested this reduction was 20.5 per cent.
2. The maximum effect occurred in thirty minutes and lasted through a period of one hour. After this a return to normal takes place rather rapidly.
3. A dose of 8 cc. gave the maximal effect.
4. The therapeutic dose recommended is 8 cc. repeated hourly.
5. This drug should be used only as a helpful adjuvant in the checking of hemorrhage.

Parathyroid Extract in Hemorrhage

BY DR. ABRAHAM CANTAROW

(By Invitation)

Calcium is essential to the normal coagulation of blood, but its administration has been comparatively unsuccessful in the treatment of hemorrhage. The probable reason for this is that in the process of coagulation it appears that the diffusible fraction of the blood calcium is involved. Since it is extremely difficult to increase the proportion of diffusible to nondiffusible calcium by the mere administration of calcium salts, a definite calcium effect cannot be consistently produced by this means. At the present time such an effect can be obtained by the use of parathyroid extract. Following the intramuscular administration of 15 to 20 units in adults, the clotting time was reduced from 5 to 65 per cent, the first effect being manifest in three hours, the maximum degree of reduction occurring in eight to twelve hours. In addition to this factor a

favorable influence upon hemorrhage is exerted, particularly in jaundice, by the production of a calcium effect in tissues and capillaries, the permeability of cell membranes being diminished.

In 347 patients with hemorrhage from various causes parathyroid extract was injected intramuscularly in doses of 15 to 20 units, repeated if necessary in thirty to thirty-six hours. The object was to obtain a sharp, transient increase in the available calcium of the blood and tissues or a series of such increases. It had been found in a previous study of pulmonary hemorrhage that the maintenance of a high calcium level increases the hemorrhagic tendency and prolongs the coagulation time.

The bleeding was completely controlled in 304 of the 347 patients observed. Although it is very difficult to ascribe cessation of hemorrhage to treatment, certain points favor this view in the present study. The association of a reduction in coagulation time with diminution in bleeding, the successful results obtained after other treatment had failed, and, in some instances, the return of bleeding following the disappearance of the calcium effect and its cessation after a second injection of parathyroid extract, all suggest that the results were in large part due to this substance.

In most instances a single injection was effective, and in none of the successful cases were more than three necessary to stop the bleeding. Good results cannot be expected in hemorrhage associated with large vessel erosions, extensive areas of tissue infection, diminished retractibility of vessels, purpura hemorrhagica and hemophilia, although the coagulation and bleeding time are reduced in most instances.

The administration of 20 units in ten hours before operation appears to be of aid to the surgeon by creating a drier field of operation and by diminishing postoperative oozing of blood and serum. This is of particular benefit in any surgical procedure upon patients with jaundice, since in this condition the hemorrhagic tendency is associated with a disturbance in the availability or utilization of calcium.

Discussion. Dr. S. MacCuen Smith said: I have used Ceanothyn very extensively since it was placed on the market, the manufacturers assuring us that it could be given in huge doses without any untoward results; indeed, we were urged to give it in 2 or more tablespoonful doses, rather frequently repeated, the results being that, many times, patients would vomit during the tonsillectomy under general anesthesia. We are,

therefore, indebted to Drs. Giordano and Wilbur for their investigations, not only in regard to the dosage, but the duration of the efficiency of the drug. As a result of their experiments we now know definitely that the drug is just as efficient in a dosage of two drams, and this will lessen the tendency to nausea and vomiting.

We are also indebted to Dr. Abraham Cantarow for his investigations into the use of parathyroid extract. I have been using Parathormone for some time, and I can, therefore, confirm. Dr. Cantarow's findings. At the Jefferson Hospital it has been our custom to give 15 units six hours before operation, although from Dr. Cantarow's studies it seems that the maximum increase in the calcium content is not reached until eight or nine hours have passed. This definite knowledge is, of course, valuable and I shall profit by its application in the future.

Dr. George Fetterolf said that we now know that the alkaloids of Ceanothus americanus will reduce the coagulation time 20 per cent. The work reported has given us the proper dosage and how often it should be used.

Dr. Nathan P. Stauffer was surprised that normal coagulation time was found to be so long. When large vessels are severed, surgical methods should be applied to control bleeding.

Presentation of Some of the Otolaryngological Material of the Mütter Museum

DR. GEORGE FETTEROLF AND DR. HARRY P. SCHENCK

PROCEEDINGS

OF THE

SECTION ON GENERAL MEDICINE

PAPERS READ DURING 1927

JANUARY 24

Dr. John H. Arnett, "Addison's Disease and Diabetes Mellitus Occurring Simultaneously."

Dr. George W. Raiziss (by invitation), "The Chemical Constitution and Therapeutic Effect of Some of the Newer Antisyphilitic Remedies."

Dr. R. Max Goepp and Dr. Helen Ingleby (by invitation), "A Case of Hanot's Cirrhosis with Autopsy."

FEBRUARY 28

Dr. H. Alexander Clarke (by invitation), "Geographical and Meteorological Factors in Hayfever."

Dr. Edward Weiss and Dr. C. J. Bucher (by invitation), "Aneurysm of the Renal Artery."

Dr. Thomas C. Stellwagen and Dr. James F. McCahey (by invitation), "Gonorrheal Arthritis in the Adult Male, Correlation of Clinical and Urological Findings: Treatment by Injection of the Seminal Vesicles."

MARCH 28

Dr. James E. Cottrell (by invitation), "The Use of Ortho-Iodoxybenzoic Acid in the Treatment of Arthritis."

Dr. Frank Walton Burge (by invitation) and Dr. John L. Laird (by invitation), "Vaccine Therapy by the Divided Injection Method. Advantages over Single Injection Method. Theoretical and Experimental Considerations."

Dr. Edward Rose (by invitation), "Adrenal Hemorrhage with Especial Reference to its Occurrence in Leukemia."

Dr. Harold W. Jones and Dr. Walter Sussman (by invitation), "Renal Glycosuria and Pentosuria. A Discussion of the Similarity of the Two Cases."

APRIL 22

Dr. Sandor Ferenczi, Budapest, Hungary (by invitation), "Psychoanalysis in Relation to Medical Practice."

SEPTEMBER 26

Dr. Robert L. Pitfield, "Tuberculous Meningitis and Tuberculoma and the Total Spinal Fluid Chlorides."

Dr. W. L. Aycock, Boston, Mass. (by invitation), "The Problem of Poliomyelitis."

OCTOBER 24

Sir John F. H. Broadbent, London, England (by invitation), "The Etiology of High Blood-pressure."

NOVEMBER 28

Dr. Clifford B. Farr, "Prefatory Remarks."

Dr. Charles W. Leuders, "Gastrointestinal Reaction to the Emotions: the Rôle of the Vegetative System."

Dr. Charles D. Gammon (by invitation), "Gastric Psychic Secretion as Modified by Emotion: an Attempt to Develop a Suitable Technic."

Dr. M. H. Jacobs (by invitation), "Some Medical Aspects of Cell Permeability."

Dr. H. C. Bazett, "Some Effects of Temperature Changes in Man."

PROCEEDINGS

OF THE

SECTION ON PUBLIC HEALTH AND INDUSTRIAL MEDICINE

PAPERS READ DURING 1927

FEBRUARY 14

Dr. Charles C. Norris (by invitation), "Prenatal Care and its Industrial Relationship."

Dr. Hugh Chaplin, New York City (by invitation), "The Relationship of Child Health to Industry."

APRIL 5

Dr. George H. Gehrmann (by invitation), "Duties and Opportunities of the Industrial Physician."

Dr. May Mayers (by invitation), "Field Investigations in Industrial Hygiene."

MAY 9

Dr. Richard M. Smith, Boston Mass. (by invitation), "Health Supervision in the Country Day School."

Dr. Walter S. Cornell, "Health Supervision in the Philadelphia Public Schools."

Dr. S. T. Nicholson, Jr., Pottstown, Pa. (by invitation), "Health Supervision in Private Preparatory Schools."

Dr. Joseph Raycroft, Princeton University (by invitation), "Health Supervision in Colleges and Universities."

NOVEMBER 4

Dr. Theodore B. Appel (by invitation), "Department Policies."

Dr. William G. Turnbull (by invitation), "Tuberculosis Work."

Dr. J. Moore Campbell (by invitation), "Communicable Diseases."

Dr. W. L. Stevenson (by invitation) "Water Purification and Sewage Disposal."

Dr. J. Bruce McCreary (by invitation), "Child Health."

PROCEEDINGS

OF THE

SECTION ON MEDICAL HISTORY

PAPERS READ DURING 1927

JANUARY 27

Dr. W. H. F. Addison (by invitation), "Centenary of the Discovery of the Mammalian Ovum."

Dr. E. G. Conklin, Princeton University (by invitation), "Heredity and Environment in Human Progress."

Dr. Joseph Walsh, "Galen's Studies at the Alexandrian School."

FEBRUARY 24

Mr. Abraham Flexner, New York (by invitation), "The Real Inside Story of the Full-time Movement in Clinical Teaching."

Major James F. Coupal, U. S. A. (by invitation), "The History of the Etiology of Disease."

Dr. Charles H. Frazier, "Medical Superstitions and Practices in China."

MARCH 24

Dr. Joseph M. Hayman, Jr., "Carl Ludwig, a Pioneer Student of Renal Function."

Dr. Thomas McCrae, "The Life of Richard Bright."

Dr. Henry A. Christian, Harvard University (by invitation), "Bright's Disease as Described by Richard Bright in Contrast to Bright's Disease of a Century Later."

OCTOBER 27

Dr. William T. Sharpless, "Dr. John Rutty of Dublin and his Spiritual Diary and Soliloquies."

Dr. Lewis Gregory Cole, New York (by invitation), "Classification of Gastric Ulcer." (With moving picture films.)

Dr. Harold W. Jones, "The Influence of Dr. James Blundell on the Development of Blood Transfusion."

NOVEMBER 18

Dr. Temple Fay, "Cranial Surgery in the Days of Ambrose Paré."

Dr. B. Franklin Royer, New York (by invitation), "The Great Sickness at Harrisburg."

Dr. Robert L. Pitfield, "Some Account of Samuel Pepy's Oculist, D'Urberville, and his Family."

Dr. Leopold Vaccaro (by invitation), "Leonardo Da Vinci, Precursor of Modern Medicine."

DECEMBER 15

Dr. William Pepper, "The Makers of the Medical School of the University of Pennsylvania."

Dr. John H. Gibbon, "The Makers of the Jefferson Medical College."

Dr. Martha Tracy, "The Makers of the Woman's Medical College."

ANNUAL REPORT OF THE LIBRARY COMMITTEE FOR 1927.

Mr. President: In accordance with the ordinances and By-Laws of the College, I herewith submit the following report of the Library Committee for the year 1927:

Total number of volumes in the Library, including the bound volumes and 22,662 unbound "Reports" and "Transactions"	154,293
Number of unbound "Theses" and "Dissertations"	29,081
Number of unbound Pamphlets	163,064
Manuscripts	431

Included in the above are 4365 volumes known as "Reserves," consisting of second copies of some of the more important periodical publications; also 2918 volumes more or less incomplete.

The duplicates, which number 3900 at this date, are not included in the above total.

The following table shows the number of volumes in the various divisions of the Library:

	Bound.	Incomplete and unbound.	Total.
General Library	110,753	2,871	113,624
Lewis Library	13,814	44	13,858
On permanent deposit:			
S. D. Gross Library	3,739	3	3,742
Library of the Obstetrical Society of Philadelphia	217	0	217
Mütter Museum*	190	0	190
			131,631

Received during the year from all sources, 5194 volumes, 7223 pamphlets and 9049 numbers of various periodicals.

Divided as follows:

	Volumes.	Pamphlets.	Journals.
General Library	3,826	7,053	8,252
Lewis Library	15		
S. D. Gross Library	18		
By purchase, General Account	537		
In exchange	798	170	797
	5,194	7,223	9,049

* The books of the Mütter Museum are not permitted to be removed from the College building.

Accessions (including 60 volumes of "Reserves"), 3749.

	Volumes.
General Library	3,715
Lewis Library	16
S. D. Gross Library	18
	3,749

Total increase in the number of volumes for the year: 3749.

Photographs received in response to requests sent out during the past year and voluntary gifts: Fellows of the College, 14; other than Fellows, 7.

Total number of portraits listed, 13,038.

The individual "Donors" for the year ending November 1, 1927, number 496; this represents 1089 distinct presentations. Each gift is duly acknowledged.

The following list shows individual gifts of twenty volumes and over; also the number of volumes presented by the various publishing houses:

	Volumes.
Mrs. John G. Clark	576
Mrs. Henry R. Wharton	300
Dr. Hunter W. McGuire	209
Dr. George E. deSchweinitz	129
Dr. Samuel W. Morton	124
Dr. Augustus A. Eshner	98
Dr. Wilmon W. Leach	87
Dr. George M. Piersol	74
Dr. Francis X. Dercum	67
Dr. O. H. Perry Pepper	41
Dr. W. W. Keen	30
Dr. Hobart A. Hare	28
Dr. Edward W. Watson	26
United States Government	24
Dr. Barton Cooke Hirst	21
Dr. Edward B. Krumbhaar	21

From the publishing houses of:

	Volumes.
P. Blakiston's Son & Co.	24
F. A. Davis Company	15
Lea & Febiger	20
J. B. Lippincott Company	21
W. B. Saunders Company	49

The Library is indebted for large gifts of pamphlets and unbound periodicals to the following donors:

Dr. H. B. Allyn
Dr. A. P. C. Ashhurst
P. Blakiston's Son & Co.
Dr. Charles W. Burr
Dr. Burton Chance
Chicago Department of Health
Dr. Francis X. Dercum
Dr. W. A. N. Dorland
Dr. G. M. Dorrance
Dr. A. A. Eshner
Dr. C. B. Farr
Dr. J. C. Gittings
Dr. R. M. Goepp
Dr. J. P. C. Griffith
Dr. S. McC. Hamill
Dr. Hobart A. Hare
Dr. A. B. Hirsh
Mr. Paul B. Hoeber
Dr. R. J. Hunter
Dr. C. F. Judson
Dr. W. W. Keen
Dr. E. B. Krumbhaar
Dr. Joseph Leidy
Dr. G. M. Marshall
Dr. Edward Martin
Dr. Charles K. Mills
National Tuberculosis Association
New York State Department of Health
Philippine Health Service
Dr. David Riesman
Rockefeller Institute and Foundation
Dr. E. C. Russell
Dr. G. E. deSchweinitz
Dr. W. J. Taylor
United States Public Health Service
Dr. Joseph Walsh
Dr. J. C. Wilson
Dr. W. Zentmayer

844 new publications were added to the Library during the past year; viz.: United States, 318; Great Britain and Colonies, 193; France, 94; Germany, 215; Italy, 4; all other countries, 20. Of these new publications, 40 were written or edited by Fellows of the College.

26 volumes were presented by the following authors or editors:

Dr. A. P. C. Ashhurst
Dr. Joseph Brassinne
Drs. P. K. Brown, W. B. Coffey, J. D. Humber
Dr. D. Bryson Delavan
Dr. Theodore Diller
Dr. W. A. N. Dorland
Dr. Wells P. Eagleton
Dr. Eldridge L. Eliason
Dr. Emile Forgue
Dr. Hobart A. Hare (Editor)
Dr. Henry R. Harrower
Miss Mary L. Hicks
Dr. Raphael Isaacs
Dr. Edward Jackson (Editor)
Dr. Franklin H. Martin
Dr. Frederick P. Millard
Drs. Henry K. Pancoast and Eugene P. Pendergrass
Drs. P. S. Pelouze and F. S. Schofield
Mr. Seymour de Ricci
Dr. David Riesman
Sir Edward A. Sharpey Schäfer
Dr. Thomas H. Shastid
Dr. Edgar Fahs Smith

2 volumes were sent by the publishers at the request of the following authors or editors:

Dr. A. P. C. Ashhurst
Dr. Ralph W. Webster

Summary of the "Funds:"

	Volumes purchased.	Cost.
Henrietta Rush Fales Baker Fund	57	$242.02
Luther S. Bent Fund	21	49.48
William T. Carter Fund	44	200.19
Gerardus Clarkson Fund	10	20.66
Francis X. Dercum Fund	55	213.70
Louis A. Duhring Fund	69	567.60
John D. Griscom Fund	118	278.67
Richard H. Harte Fund	6	63.73
Charles T. Hunter Fund	4	17.56
William F. Jenks Fund	94	372.53
Oliver A. Judson Fund	13	64.39
William V. and John M. Keating Fund	15	51.79
William W. Keen Fund	63	299.03
Library Endowment Fund	186	912.69
Horace Magee Memorial Fund	181	926.96
J. Ewing Mears Fund	58	373.88
Charles K. Mills Fund	...	.....
Weir Mitchell Fund	51	324.69
E. E. Montgomery Fund	31	217.37
John H. Musser Fund	10	13.74
Elizabeth K. Newcomet Fund	21	68.95
William F. Norris Fund	33	158.58
Charles A. Oliver Fund	7	57.66
Philadelphia Medical Society Fund	7	33.73
Lewis Rodman Fund	48	238.44
Douglas Stockton Warren Fund	38	133.89
John F. Weightman Fund	18	39.81
J. William White Fund	26	225.82
Caspar Wistar Fund	72	257.19
	1,356	$6,424.75

Special accounts:

	Volumes purchased.	Cost.
Fund for completing files of journals	79	$429.21
Fund for rare and valuable books	39	479.59
S. D. Gross Library Account	14	149.83
From the General Account—by purchase	537	3,241.65

	Expended.
Catalogue Endowment Fund—on account of salaries	$46.50
Louis A. Duhring Fund—general purposes of the Library	12,331.12
Morris Longstreth Library Fund—on account of salaries	1,107.00
George B. Wood Fund—Library supplies, stationery, etc.	369.23

Number of volumes bound, 2601.

	Visitors.	Fellows.	Total.
Use of the Library	5,850	1,511	7,361

The Library has been kept open two evenings each week and on the five minor legal holidays for the same period of time as during the previous year:

Evenings.	Visitors.	Fellows.	Total.
75	854	134	988

Legal holidays.	Visitors.	Fellows.	Total.
5	77	18	95

These figures are included in the total given for the year.

	1927.	1926.	Decrease.
Number of volumes consulted in the Library	25,363	29,862	4,499

The number of books "consulted in the Library" includes only those supplied on demand. Readers have access to the bound volumes of periodicals and works of reference kept on the shelves in the Reading Room, and the Fellows of the College have access to the Book Stacks. There are, therefore, many volumes consulted of which no accurate record can be kept.

	1927.	1926.	Increase.
Number of volumes taken out:	4,449	3,559	890

Use of Study Rooms: Average number of rooms in use throughout the year, 6. Greatest number in use at one time, 10. Daily average of the number of volumes in use in these rooms, 150.

	Works.	Volumes.	Cards written.	Printed cards revised.
Cataloguing . . .	1,024	1,050	3,887	1,991

All books added to the shelves during the past year and 181 of the more important pamphlets have been catalogued and shelf-listed; and all bound volumes, including periodicals, transactions and reports, have been accessioned.

Revision of the Catalogue: 6177 cards have been revised, rewritten and filed.

1929 unbound pamphlets and reprints have been subject-headed and arranged alphabetically by subject and author under the

subject. 5733 pamphlets and reprints have been bound in 297 volumes; one subject or division of a subject to a volume. Each volume has a table of contents arranged alphabetically by author. A catalogue card will be filed under each author's name, and one subject card for each volume.

We have listed November 1, 1927, 1325 current periodical publications including "Transactions" and "Reports," obtained through the following sources:

	American.	Foreign.
Endowment Funds	86	359
By purchase from General Account	39	190
In exchange	68	136
Editors	91	28
Publishers	82	21
Through the courtesy of the Editor of the *Therapeutic Gazette*, current numbers are received at stated intervals	42	43
	408	777
Reports, not included in the above	112	28
	520	805

The actual number of periodicals received during the year, including new subscriptions (2 American, 12 foreign) total 1185.

The following is a list of the foreign schools of medicine with which we exchange publications at the present time:

University of Amsterdam	University of Königsberg
" Basel	" Lausanne
" Berlin	" Leiden
" Bonn	" Leipzig
" Breslau	" Liège
" Erlangen	" Lund
" Geneva	" Marburg
" Giessen	" Rostock
" Göttingen	" Strassburg
" Halle	" Upsala
" Heidelberg	" Würzburg
Faculty of Medicine of Paris	Faculty of Medicine of Toulouse

During the past year we have heard from Amsterdam, Berlin, Bern, Breslau, Erlangen, Giessen, Göttingen, Halle, Heidelberg, Kiel, Königsberg, Leipzig, Liège, Lund, Marburg, Paris and Utrecht.

1537 dissertations and 1147 theses have been added to our collection.

We have received in cash from the sale of duplicates for the current year ending November 1, 1927, $103.00.

We have distributed books and journals on exchange account to the following:

Boston Medical Library
New York Academy of Medicine

We have received exchanges from:

Boston Medical Library
Medical Library Association
College of Physicians and Surgeons, New York

With the aid received from exchanges and purchases made with funds appropriated by the Library Committee we have been able to complete the files of the following journals:

Archiv für klinische Chirurgie, Berlin
Arte Medica, Milano
Braithwaite's Retrospect of Medicine, London
Deutsche Zeitschrift für Thiermedicin und vergleichende Pathologie, Leipzig
Zodiacus Medico-Gallicus, Genevae

Amount of fines collected for the current year ending November 1, 1927, $87.75.

List of rare medical books and works of special interest received during the year (these rare books have been acquired without in any way interfering with the purchase of current medical publications):

INCUNABULA

(Total number of incunabula at this date, 376 Titles, 360 volumes.)

Auctoritates Aristotelis.*** Propositiones Aristotelis. [Venetiis, circa 1490–1495.]

Presented by Mr. Wilfrid M. Voynich.

Gratia dei Esculanus s. Asculanus, Ordin. Praedic. Commentaria in artem veterem Aristotelis. Venetiis, Octavianus Scotus per Bonetum Locatellum, Presbyter Bergomensis, 1491. [Hain 7874.]

Fund for Rare Books.

Purliliarum, Iacobus Comes. [De generosa liberorum educatione.] Tarvisii, Gerardus de Lisa de Flandria, 1492.

Presented by Edward B. Krumbhaar, M.D.

de Saliceto, Guilielmus Medic. Placent. [De salute corporis cum Iohannis de Turrecremata tractatu de salute animae.] Lipsiae, Arnoldus de Colonia, 1495. [Hain *14152.]

[*Bound with a leaf from an old German book.*]

Fund for Rare Books.

Salis s. Trouamala, Baptista de. Summa casuum conscientiae dicta Rosella seu Baptistiana. Venetiis, Georgius de Rivabenius, Arrivabenus Mantuanus, 1495. [Hain 14183.]

Fund for Rare Books.

Seneca, L. Annaeus. Opera philo[so]phica et epistolae. Venetiis, Bernardinus de Choris, Cremonensis, 1492. [Hain-Cop. *14594.]

Fund for Rare Books.

Theobaldus Episcopus. [Physiologus de naturis duodecim animalium.] Coloniae, Henricus Quentell Coloniensis [circa 1490]. [Hain-Cop. *15471.]

Photostat copy only (through the courtesy of Mr. Wilfrid M. Voynich).

Photostat Fund.

Works of Special Interest

Belloste [Augustin]. Le chirurgien d'hôpital.*** Paris, Chez Laurent d'Houry, 1696.

Fund for Rare Books.

Berrettinus, Petrus. Tabulae anatomicae, a Gajetano Petriolo Romano. Notis illustratae. Romae, Ex. typ. Antonii de Rubeis apud Pantheon in via Seminarii Romani, 1741.

Fund for Rare Books.

Borelli, Giovanni Alfonso. De vi percvssionis liber.*** Bononiae, ex tip. Iacobi Montij, 1667.

Fund for Rare Books.

Browne, Sir Thomas. Pseudodoxia epidemica; or, enquiries into very many received tenents, and commonly presumed truths. With marginal observations.*** 5 ed. London, Printed for the assigns of Edward Dod, 1669.

Fund for Rare Books.

[Browne, Sir Thomas.] Religio medici, with annotations never before published, upon all the obscure passages therein, also, observations by Sir Kenelm Digby, now newly added. 5 ed. London, Milbourn, 1659.

Fund for Rare Books.

Debates in Parliament respecting the Jennerian discovery, including the late debate on the further grant of twenty thousand pounds to Dr. Jenner. Together with the report of the Royal college of physicians of London on the vaccine inoculation. With introductory remarks, by Charles Murray. London, Phillips, 1808.

Fund for Rare Books.

Delphinus, Julius. In III Galeni artis medicinalibus lib. explanatio.*** Venetiis, Camotius, 1557.

Fund for Rare Books.

Deschamps, l'Abbé. Cours élémentaire d'éducation des sourds et muets. Paris, Debure, 1779.

Fund for Rare Books.

de Diemerbroeck, Isbrandus. Opera omnia, anatomica et medic.*** Nunc simul collecta, et diligenter recognita per Timannum de Diemerbroeck, Isb. fil. 2 ed. Patauij, ex typographia Petri Mariae Frambotti, 1688. [*Engraved title page.*]

Fund for Rare Books.

Dissertationum lvdicrarum et amoenitatvm. Scriptores varij. Editio nova. Lvgd. Batavor., apud Franciscum Hegerum, 1644.

Fund for Rare Books.

Dullaart, J[an]. Alexander de Medicis, 't bedrooge betrouwen. Treurspel. In Duitsche vaarzen gestelt door. t'Amsterdam, Bouman, 1663.

Fund for Rare Books.

Fialetti, Odoardo. Il uero modo et ordine per dissegnar tutte le parti et membra del corpo humano. Venetia, Sadeler, 1608.

Fund for Rare Books.

Fuchs, Leonhard. De sanandis totius humani corporis malis. London, apud Ioannem Frellonium, 1547.

In Exchange.

[Gibson, E.] Causes of the discontent in relation to the plague. London, Roberts, 1721.

Fund for Rare Books.

History of the damnable life and deserved death of Doctor John Faustus 1592, together with the second report of Faustus containing his appearance and the deeds of Wagner, both modernized and edited by William Rose.*** London, Routledge, 1925.

Fund for Rare Books.

Ibn Djezla, Abul Hassan Ali ben Issa. Taqwim al Abdan. Damascus, Rashid Pasha, Wakil of Ibn Rashid, Amir of Jauf, 1333. [*Reproduced from original manuscript, 1927.*]

Fund for Rare Books.

Illingworth, J. A. Just narrative, or, account of the man whose hands and legs rotted off; in the parish of Kings-Swinford, in Staffordshire, where he died, June 21, 1677.*** London, Browne, 1678.

Fund for Rare Books.

Jamblichus. De mysteriis Aegyptorvm, Chaldaeorum, Assyiorum. Lvgdvni, apud Ioan. Tornaestum, 1649.

Fund for Rare Books.

Maynwaring, Everard. Praxis medicorum antiqva et nova; the ancient and modern practice of physick examined, stated, and compared.*** London, Printed by J. M. and are to be sold by T. Archer, 1671.

Fund for Rare Books.

Mesue Damascenus, Joannes. Canones universales.*** Lugd[uni], Antonius du Ry, impensis J. and F. de Giunta, 1531.

This little medical handbook enjoyed great popularity from the later XVth century until the latter half of the XVIth century. This edition of it is rare.

Fund for Rare Books.

The notorious impostor, or the history of the life of William Morrell, alias Bowyer.*** Sometime of Banbury, chirurgeon. London, Roper, 1692.

Fund for Rare Books.

[Poole, Robert.] A physical vade mecum; or, fifth gift. Wherein is contain'd the dispensatory of St. Thomas's hospital, with a catalogue of diseases, and the method of their cure prescrib'd in the said hospital.*** London, Printed for, and sold by E. Duncomb, in Duck-Lane, Little Britain, 1741.

Fund for Rare Books.

Rabelais, François. Les oeuvres. 2 volumes in 1. [Amsterdam, Elsevier] 1666.

Fund for Rare Books.

Redi, Francesco. Opere. Edizione Veneta seconda ricorretta. 6 volumes. Venezia, appresso gl'Eredi Hertz, 1728–1742.

Fund for Rare Books.

Riverius, Lazarus. Institvtiones medicae, in qvinque libros distinctae quibus totidem medicinae partes, physiologia, pathologia, semeiotice, hygiene, et therapeutice dilucidè explicantur.*** Lvgdvni, Cellier, 1656.

Fund for Rare Books.

Rolfinck, Werner. Ordo et methodus medicinae specialis commentatoriae, *** dolorem capitia, ad normam veterum et novorum dogmatum proposita.*** Jenae, Johannes Nisius, 1671.

Fund for Rare Books.

Vegetius Renatus, Publicus. Artis veterinariae. Basileae [Emmeus, 1528]. *First printed book on veterinary medicine. Very rare edition.*

Fund for Rare Books.

Wedel, Georg Wolffgang. Opiologia. Jenae, Sumptibus Johannis Bielkii Bibliop. typ. Viduae Samuelis Krebsii, 1682.

Fund for Rare Books.

Zorn, Bartholomaeus. Theses medicae. De venaesectionis necessitate.*** Altdorfi, e typ. Acad. Hagenianâ, 1661.

Fund for Rare Books.

Manuscripts

Darrach, William. Sec'y. Medical Prayer-Meeting, Committee on prisons. Report. [Phila.] 1828.

Presented by Herbert Fox, M.D.

de Novellino [Philippus]. Institutiones medicinae. sive fundamenta maecanicis (sic) legibus innixa. Naples, 1729.

Fund for Rare Books.

Other Interesting Additions

Holograph "Directions for the Treatment of Ague and Fever" written by A. C. Logan, Stenton, October 10, 1813.

Presented by Burton Chance, M.D.

A Gavel given to the late Doctor John G. Clark by the Medical Club of Philadelphia.

Presented by Mrs. John G. Clark.

Four diplomas of William Wood Gerhard, M.D., from the American Philosophical Society (two); Dickinson College and the University of Pennsylvania.

Presented by Miss Anne Gerhard Maris.

We have also received during the past year a number of interesting and more or less valuable autograph letters.

Use of the Photostat: During the past year there have been furnished to Fellows of the College 300 prints, and to other than Fellows, on application, 162 prints. These outside applications include requests from Atlanta, Georgia; Boston; Cleveland; Montgomery, Alabama; New York; Omaha, Nebraska; Philadelphia; and Rochester, Minnesota.

The work for the Library has been continued as heretofore according to the instructions of the Library Committee. 3548 large and small prints have been made; 14 volumes of incunabula completed as well as various portraits and manuscripts.

Up to the present time there have been made photostat reproductions of 254 incunabula.

There has been an increase to the principal of the Library Endowment Funds through change in investments during the past year as follows: Louis A. Duhring Fund, $2820.57; Oliver A. Judson Fund, $139.91; William W. Keen Fund, $429.56; J. Ewing Mears Fund, $290.44; John H. Musser Fund, $18.59; Caspar Wistar Fund, $139.91 and the George B. Wood Fund, $121.53. A complete list of all the Funds and the expenditures for the year is given in a previous part of this report. All additional expenses, salaries, binding, etc., have to governed according to the funds appropriated by the College to the Library Committee annually.

The rearrangement of periodicals, reports and transactions in the Stacks into one alphabetical arrangement, instead of by countries, has been completed; this work was necessarily slow and required great care so as not to interfere with the use of the Library.

The figures reported show that the affairs of the Library have progressed in a satisfactory manner; in the number of new publications received as well as the receipt of a number of valuable curios; in the use of the Library and the number of books taken out; the amount of binding completed, and the completion of the rearrangement of the entire contents of the Book Stack. The catalogue and revision of the catalogue has advanced and 5733 pamphlets and reprints have been bound in 297 volumes; a single subject to the volume.

Library Committee,

Francis X. Dercum, M.D.,
Chairman.

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